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A. V. M. A. Meeting

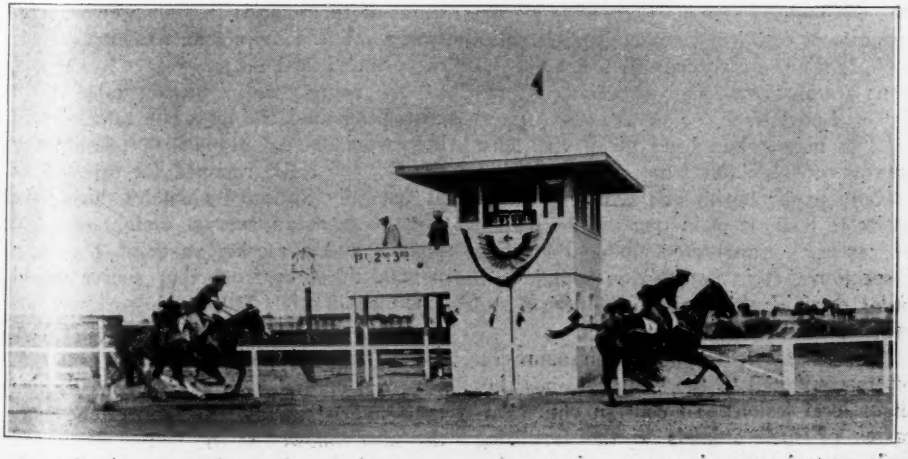
Dr. M. Scherago, Chairman of the Publicity Committee, states that those in charge of local arrangements for the coming meeting of the A. V. M. A., which will be held in Lexington, Ky., August 17th to 20th, are exceedingly anxious to have as large an attendance of veterinarians as possible.

Besides the usual business of such meetings, the local Committee on Arrangements has prepared a very interesting program, consisting of clinics and demonstrations as well as enjoyable social features. They are also planning to take all of the visiting veterinarians and their friends on a trip to the various breeding farms in the Blue Grass region and to show them some of the best thoroughbred horses in the world.

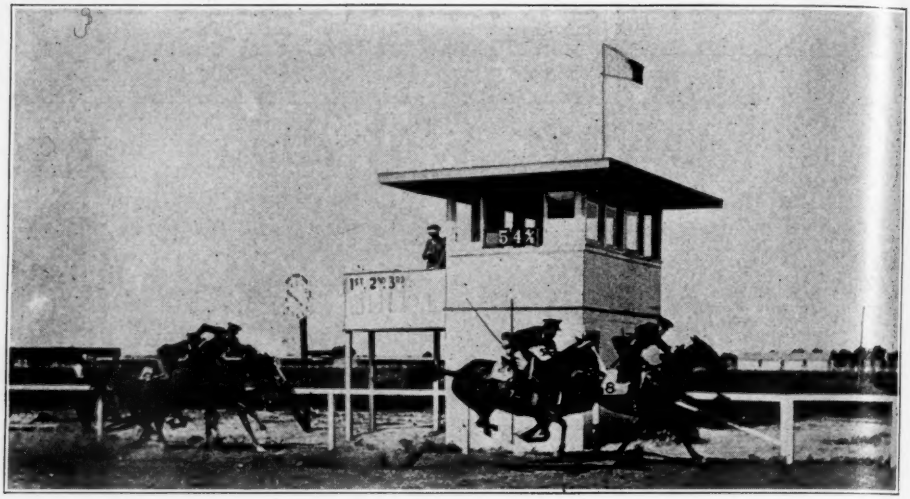
The roads coming into Lexington are all reported in excellent condition, and the Blue Grass city is particularly fortunate in being located at the intersection of five great national highways—the National Midland Trail, the Dixie Highway, the Dixie Route A, the Boone Way and the Appalachian Way.

It is claimed that a tour through Kentucky to the beautiful Blue Grass region is something worth while traveling for in itself. For those who expect to tour to Lexington, there is an excellent free tourist camp at Joyland Park. This camp is equipped with city water, electric lights, toilets, shade trees, benches, park seats, tables, etc. In the park itself there is a good restaurant and a dance hall, where music is furnished by some of the best orchestras in the country. Opposite the camp is a grocery store; and interurban service into Lexington as well as bus service, makes this camp an easy approach to the city, which is only three miles away.

Dr. Scherago states: "Everybody in Lexington is being primed for the big event, and we are just 'rarin'' to show the veterinarians the best time they have ever had in their lives. That is why we are so anxious that no one miss this opportunity to partake of the famous Kentucky hospitality."



Captain R. Payne McComb, V. C., U. S. Army, with his horse "Easter Day," winning the Chief of Cavalry Handicap at Juarez, Mexico, May 2, 1926.



Captain R. Payne McComb, V. C., U. S. Army, riding his horse "Easter Day" to victory at the Marfa Military Handicap, Juarez, Mexico, May 16, 1926.

DISPOSAL OF TUBERCULOUS ANIMALS

In a recent circular by Dr. J. R. Mohler, Chief of the Bureau of Animal Industry, is discussed the policy of using portions of tuberculous animals for food, in view of the fact that some people have urged the total destruction of the carcasses of all reactors. The following excerpts are of special interest in this connection:

"Carcasses showing evidence of generalized tuberculosis may be used only for such purposes as fertilizer or other inedible products. Portions of animals in which the lesions are small or localized may be passed for food purposes.

"The matter has been given the most careful consideration from the standpoints of both public health and economy, and I desire to make it plain that the disposition of carcasses slaughtered under the Meat Inspection Act is founded on sound principles, established by eminent physicians and veterinarians.

"On postmortem examination many reactors consigned to the shambles show but slight local lesions. To consign entire carcasses of such animals to the fertilizing tank, or to bury such carcasses in trenches as was recently suggested, would be like throwing entire bunches of grapes into the garbage can because one or two small grapes on a bunch were unsound. It would be a willful waste of wholesome food.

"The committing of all tuberculous animals to the trench or fertilizer tank would impose on the public a far greater economic loss than most people believe until they look carefully into the matter. It is estimated that the 7,039,724 swine 'retained' on account of tuberculosis in 1925 would aggregate a total of more than a billion pounds of pork and lard. At an average live weight of 200 pounds and a market value of 10 cents per pound, the aggregate value of such hogs would exceed $140,000,000. Estimating the 322,925 cattle retained for tuberculosis at federally inspected abattoirs during the same year at an average weight of 1,000 pounds and a market value of 7 cents a pound, the loss from that source would amount to $22,604,750. The loss of both cattle and hogs would amount to more than $162,000,000 annually.

"I am sure that after due consideration unprejudiced minds will approve the practice now followed in the United States and in practically all foreign countries."

STUDY OF FOOT-AND-MOUTH DISEASE ENDS

The foot-and-mouth disease commission sent to Europe to study the research being done there on the problem has returned after more than a year's stay. The commission, which consists of Dr. Peter K. Olitsky of the Rockefeller Institute for Medical Research, Dr. Jacob Traum of the University of California, and Dr. Harry W. Schoening of the Federal Bureau of Animal Industry, is now engaged in writing its report of the survey.

After studying the research work and the control measures employed in France, Germany, England, Denmark, Sweden, Holland, Belgium, Switzerland, Austria, Hungary, and Italy, the commission established itself at the Institut d' Hygiene, Strasbourg, and later also at the Laboratoire National de Recherches, Alfort, France. Experimental studies were made at these institutions on the causal agent of foot-and-mouth disease from its physical, chemical, and biological aspects. Particular attention was given to certain phases of the problem bearing on methods of control of the disease used in this country.

European investigators and officials gave the fullest cooperation and expressed their approval of the commission's work. It is hoped that this effort will result in solving some of the problems of this disease which causes such great economic losses all over the world. The scientists returned at this time because the appropriation for the study lapsed July 1st.

Insulin in a crystalline form has been produced by Abel and his co-workers at Johns Hopkins University. The purity of the crystalline insulin is indicated by the fact one milligram of this product is equal to 100 clinical units of the solution used in medical practice.

ARMY OFFICERS TO ATTEND A. V. M. A. MEETING

In view of the fact that many of the veterinary officers of the regular army are members of the A. V. M. A., the Surgeon General has stated that it is most important that these officers be allowed to attend the annual meetings of their association whenever possible. All Corps Area commanders have been authorized to permit officers of the Veterinary Corps under their jurisdiction to attend the meeting at Lexington without expense to the government, provided the services of such officers can be spared.

Captain R. Payne McComb, V. C., U. S. Army, on "Easter Day," in the winning ring immediately after beating the Cavalry in "The Chief of Cavalry Handicap," May 2nd. Private Rose (Veterinary Service) at horse's head.

BOWMAN ABORTION REMEDY CONDEMNED BY COURT

So-called remedies for contagious abortion in cattle and swine received another blow when representatives of the United States Department of Agriculture exposed the absurdity of the claims made by the Erick Bowman Remedy Company of Owatonna, Minnesota, in a trial before the United States District Court in Cleveland, Ohio, May 5. The court recently handed down its opinion declaring the company's product misbranded and ordered the quantity before the court destroyed. The "Remedy" was found to consist mainly of brown sugar and wheat bran.

First knowledge concerning this particular medicine came to the department in 1921, when Erick Bowman, a farmer, then living near Claremont, Minnesota, asked the department to purchase the secret formula from him for a preparation which he claimed would prevent and cure contagious abortion. Failing in his attempt to sell the idea he began to manufacture and sell the mixture to farmers in his neighborhood. He was able to secure favorable testimonials from many of them. By 1923 a company had been organized and incorporated as the Erick Bowman Remedy Company. An extensive campaign was then inaugurated by the company for the sale of Bowman's Abortion Remedy throughout the United States.

An investigation was then undertaken by the department. Repeated analyses showed that the preparation consisted of about 85 per cent brown sugar with which about 15 per cent of ground wheat bran was mixed. The results of these analyses were in accord with those obtained by officials in various states. Even though present scientific knowledge of the disease and its cause precluded any possibility that this mixture could be of any value as a preventive or cure for contagious abortion and allied conditions, further investigations were undertaken under farm conditions with laboratory controls. Definite proof was thus obtained that Bowman's Abortion Remedy had no influence in destroying the germs of abortion or in protecting cattle from becoming infected and losing their calves.

In April, 1925, the Bowman Company was notified that their product was considered to be misbranded within the meaning of the Federal Food and Drugs Act. Shortly after receiving this warning the company attempted to evade the spirit of the law by covering a part of the label, leaving accessible that portion giving directions for the use of the mixture. Following this action the department seized a number of interstate shipments at various points throughout the country. The trial was instituted for the indemnification and forfeiture of a particularly large shipment seized at Ravenna, Ohio, which was intended for further distribution from that point to individual purchasers in the surrounding states. The so-called remedy was found misbranded because of the false and fraudulent claims made for it and the court ordered it destroyed by the United States Marshal.

The department was represented at the trial by Dr. B. T. Woodward, Cooperative Adviser to the Bureau of Chemistry; Dr. W. E. Cotton, Assistant Superintendent of the Animal Disease Experiment Station; Dr. J. S. Jamieson and Dr. W. H. Callahan, Medical Officers, and C. K. Glycart, Chemical Analyst. Further testimony in behalf of the Government's stand was offered by such eminent authorities in the veterinary profession as Dr. R. A. Craig of Purdue University, Dr. M. F. Barnes of the Pennsylvania State Bureau of Animal Industry, Dr. Ward Giltner and Dr. I. F. Huddleson of the Michigan Agricultural College, Dr. L. W. Goss of the Ohio State University, and Dr. W. L. Boyd and Dr. C. P. Fitch of the University of Minnesota. It was the consensus of opinion of all of these authorities that there is no known drug or combination of drugs which is of any value whatsoever in the prevention, or cure of abortion disease of cattle or swine and that certainly sugar and bran could not do it.

Five dollars for 9½ pounds of a sugar and bran mixture is an exorbitant price to pay for these materials, but that is what the buyers of the Bowman Remedy paid.

The Agglutination Test for Brucella Abortus Infection*

CHARLES M. CARPENTER, Ithaca, N. Y.

1. The agglutination test for infectious abortion demonstrates the presence of Brucella abortus agglutinins (antibodies) that may be found in the blood serum of an animal.

2. Complete agglutination of a salt suspension of Brucella abortus by blood serum from an animal diluted 1-60 is evidence of the infection. A much higher serum titre such as 1:540 or 1:1620 indicates, as a rule, a recent infection.

3. A positive blood test does not necessarily mean that a pregnant animal will abort or have retained placenta. A heifer in her first pregnancy or adult that has never aborted is more liable to show these symptoms than an animal that has aborted previously. A positive test denotes that the animal is harboring the infection. If she is non-pregnant the Bang organism can, as a rule, be found in the udder, spleen or lymph glands.

4. The blood test will detect carriers and spreaders of Brucella abortus that may be calving apparently normally.

5. The blood test will detect infected animals that do not abort but have only enough placentitis due to Brucella abortus to cause retained placenta.

6. A sample of blood drawn from an animal on the day of abortion may show only a few or no antibodies.

7. As a rule, the antibodies or agglutinins are present in the blood serum two weeks after infection, but this depends upon the amount of infection to which the animal has been exposed and her individual susceptibility.

8. The agglutination test upon the blood of calves and very young heifers is unsatisfactory and, as a rule, not necessary.

9. A positive blood test for abortus infection may remain throughout several pregnancies due, undoubtedly, to udder infection. A large percentage of infected animals remain infected throughout their lives.

10. Successive blood tests may be made upon any animal at any interval.

11. A satisfactory interpretation of a blood test can be made only when the breeding history of the animal is known.

12. If an animal has been injected with a living or dead culture of Brucella abortus the interpretation of the results of the agglutination test cannot be relied upon.

Practical Application

1. One agglutination test upon the animals comprising a herd determines the number of infected animals. A plan to follow in eliminating the disease depends upon the per cent of animals infected.

2. Animals reacting positively to the test should be segregated, if possible, and a pregnant animal should be observed for premonitory symptoms of abortion.

3. Blood test should be made 60 days apart until two or three negative tests have been made. Two tests a year thereafter are sufficient.

4. Newly purchased animals should not be placed with negatively reacting animals until a blood test has been made showing them free from infection. A second blood test six weeks later is advisable.

5. Calves may be allowed to nurse infected dams and to associate with them until three months before breeding when they should be placed with the negative group after a negative blood test. A blood test early in their first pregnancy is desirable.

6. A blood test should be made upon bulls to be used for breeding females negative to the test.

*Synopsis of paper presented at the New York State Association meeting, June 29, 1926.

7. Success with the agglutination test in controlling this disease depends upon the early recognition of the infection.

8. When abortions occur in a herd which shows a negative test for the Bang infection, undoubtedly they are due to micro-organisms other than Brucella abortus. A bacteriological examination of the fetuses and placentas should be made to determine the causative agent.

Some Gastric Phenomena of Ruminants

L. W. WYNN-LLOYD, Carnarvon, Wales

In the act of rumination the following organs play the premier part:—the esophagus, the gastric groove, the rumen and reticulum.

Very little appears to be known of the special function of the reticulum. Sisson has pointed out that its strong muscular wall would indicate that it is adapted to perform some unusual contractile effort. Ellenberger has noted that the entire sac contracts at once and that it does so with considerable energy and rapidity, after the manner of striped muscle.

The theories are obviously untenable that the particular functions of the reticulum are:—

(1) To retain foreign bodies and indigestible substances and

(2) To act as a water receptacle to supply the rumen.

Foreign bodies are found most commonly in this compartment because of its position immediately below the esophageal opening. Gravitation deposits them there and when the viscus contracts they are for the most part retained by the aid of the deep polyhedral cells of the reticular lining. The small capacity of the reticulum (3½ pints) rules it out as a necessary reservoir of water for the rumenal contents.

The opinion expressed by Ellenberger, quoted by Smith—" that the recticulum regulates the passage of ingesta from the rumen to the omasum" is also difficult to comprehend, inasmuch as there is no conclusive evidence that such passage exists at all except perhaps of fluids and some particles of ingesta in suspension or solution. In the case of fistula of the reticulum opening externally in the neighborhood of the xyphoid cartilage, we observed:

(1) That the squirting of the fluid ingesta through the opening synchronized with the abdominal contractions during rumination.

(2) That these muscular contractions were much more vigorous than usual.

(3) That rumination was apparently performed with great difficulty. These phenomena would tend to show:

(a) That the rumen and reticulum contract simultaneously.

(b) That the reticular contraction was incomplete owing to adhesions between its fundus and the abdominal floor.

(c) That greater abdominal and rumenal effort was needed to overcome this disability.

It would therefore seem that the reticulum plays no mean part in the process of normal rumination.

The part played by the gastric groove in the act of rumination is not very clear. The generally accepted theory that its lips part, the rumen contracts and brings the ingesta into the groove and that reverse peristalsis in the esophagus completes the transit of the bolus to the mouth, is open to grave doubt.

Flourens sutured the lips of the groove and claimed that rumination still took place. He, however, did not state the distance between the sutures nor whether the act was

performed in a normal manner as to the size of the bolus and number of masticulatory cuts. If the sutures were placed at wide intervals, it would be quite possible that a small bolus could be expressed between them or through the somewhat elastic extremities of the lips. His experiment was, therefore, inconclusive. To deny that the groove plays any part in the act seems unreasonable in view of its position and anatomical arrangement. If, with the arm through the surgical opening in the flank, a handful of ingesta is held against the lips of the groove, the latter move in a spasmodic fashion. If the lips are separated and a portion of ingesta inserted between them, the same clonic spasm is noted.

The writer is inclined to think that the phenomena of rumination occur in the following order:

(1) Contraction of the abdominal muscles.

(2) Contraction of the rumen and reticulum—the latter contracting very vigorously and quickly, thus pressing the semi-fluid ingesta into the gastric groove whose lips open automatically to receive it.

We have observed that the administration of aloes in solution, turpentine and creosote, will often cause suspension of the act for several hours in animals which were ruminating up to the time of receiving the dose.

Begg has reported the same result after prescribing magnesium sulphate and gamboge to healthy cows due to calve with the view to ward off an attack of parturient paresis.

The outward manifestations of cudding have been so well described by various writers—recently by Mr. Hugh Begg, F. R. C. V. S., whose keen observation has enabled him to place before the clinician a very clear exposition of the various phases of the process, that it is not necessary to repeat them here.

Normal rumination has been compared with vomition; indeed, it has been termed physiological vomiting. The two acts differ in some important points; indeed, they have very little in common, except that in each there is contraction of the abdominal muscles, and the reversed upward movement of aliment and relaxation of the cardia.

The vomiting centre in the bovine medulla is apparently not highly developed, for although this animal possesses all the anatomical attributes which would facilitate the act, its occurrence is comparatively uncommon.

True vomition should not be confused with so-called "dropping of the cud" which may be due to diseased or loose teeth, actinomycosis or other lesions of the buccal cavity, to "Bell's palsy," a result of pathological or traumatic injury to the nerve, etc.

Irritation of the fauces will not induce vomition in the bovine as in man and the writer is inclined to believe that the vomiting reflex in the ox is related to the gastric groove.

Apart from the generally accepted causes of vomition—the irritation of acrid foodstuffs and poisons, pressure on the vagus by neoplasms and enlarged lymph glands, growths and traumatisms involving the brain, etc., the writer has encountered a common cause of vomition which implicates the gastric groove.

It should here be explained that our field is, for the most part, confined to "scrub" (non-pedigree) cattle whose winter food consists chiefly of hay grown on old pastures and containing a proportion of rushes and bracken. It thus includes much indigestible fibre.

Instances of vomition, unaccompanied by digestive disturbance are here comparatively common without any very apparent cause. The animal ruminates indifferently, there may be two or more abdominal contractions before the bolus is brought up. Suddenly a varying quantity of fluid ingesta is emitted by way of the mouth and nostrils. This occurs at intervals and if not relieved the animal begins to suffer from malnutrition.

Searching for a possible cause after having performed gastrotomy, a dry bolus of fodder was found fairly firmly lodged in the gastric groove. Thereafter basing our remedial treatment of other apparently similar cases on what we had discovered, we adopted the expedient of flushing the groove by

means of a gum-fibre stomach syphon. The result in every instance has been most gratifying. Only in one case was it necessary to repeat the procedure and that a month later when a recurrence took place apparently due to the same cause. That such interference with the gastric groove acts as a cause of vomition seems also proved by a few unrelieved cases wherein autopsy has revealed foreign bodies embedded in the groove or at the reticulo-omasal opening. Further, the phenomenon, or an attempt at it (gagging) can be induced in many cases by passing the same tube into the rumen and alternately pressing it downwards and withdrawing it quickly. Another interesting point in regard to this unique structure is its influence on the production of tympany. Its cardiac end normally lies below the eighth thoracic vertebra. When the rumen is full and the diaphragm relaxed, the cardia approximates the fifth thoracic vertebra and it is situated a few inches below the ninth vertebra when the diaphragm is contracted and the rumen comparatively empty.

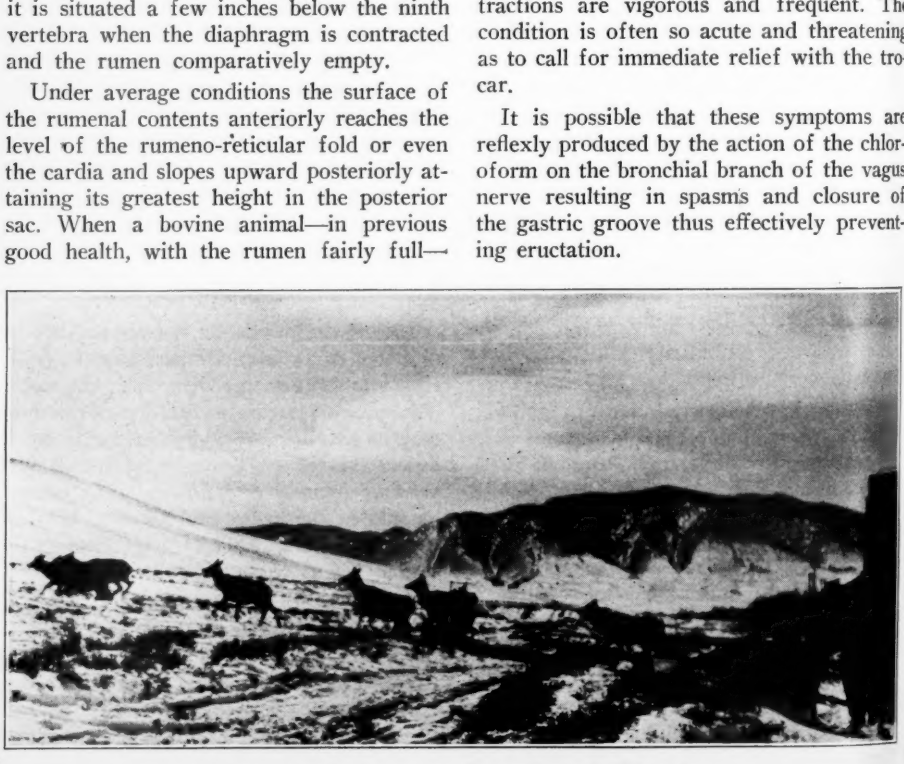
Under average conditions the surface of the rumenal contents anteriorly reaches the level of the rumeno-reticular fold or even the cardia and slopes upward posteriorly attaining its greatest height in the posterior sac. When a bovine animal—in previous good health, with the rumen fairly full—suddenly assumes the lateral recumbent position, such as happens in parturient paresis, dystocia, etc., the relative position of the surface of the ingesta is changed. The food mass presses against the lips of the groove, thus inhibiting more or less effectively the normal eructation of gases generated in the rumen. If the animal is again placed in the natural posture on the sternum, the anterior level of the ingesta is lowered, the gastric groove is freed, eructation is established and the tympany speedily disappears.

It is interesting to note the occasional influence of intra-tracheal injection of chloroform for the treatment of verminous bronchitis in setting up tympany.

If the animals have been fed prior to the injection a large proportion of them will immediately show signs of dyspnea and develop alarming tympany.

The rumen is set in active motion, its contractions are vigorous and frequent. The condition is often so acute and threatening as to call for immediate relief with the trocar.

It is possible that these symptoms are reflexly produced by the action of the chloroform on the bronchial branch of the vagus nerve resulting in spasms and closure of the gastric groove thus effectively preventing eructation.



Unloading elk at game reserve, Laramie, Wyoming.

ARKANSAS STATE AGRICULTURAL COLLEGE JUL 31 1926 LIBRARY

The Treatment of Texas Fever

B. T. HOPKINS, Lamont, Fla.

I am writing this article not because I consider myself an authority on the disease, but because I hope the experiences I have had may help some other practitioner to save a few lives that would otherwise perish from this malady, perhaps the most fatal of all bovine diseases.

It is an acknowledged fact that there is no known specific remedy that will cure Texas fever. Neither is there any preventive remedy except to keep ticks off the cattle. Ticks are the causative agent in bringing about Texas fever. In localities where the ticks exist, no power on earth can prevent cattle brought from a tick-free territory from getting the fever. Once an animal has had the fever and recovered, it is immune only so long as it carries ticks. But if entirely freed from ticks and again subjected to them, say ten or twelve months later, it will have the fever again. Cattle under fifteen months old seem to have a tolerance for the fever that keeps the death rate low at and below this age. Cattle that have had the fever and recovered are carriers of the infection. Mature ticks dropping off such animals are infected, and seed ticks hatched from these will in turn infect susceptible cows.

If left alone more than 90 per cent of cattle taken with the fever will die. On the other hand, if persistent, consistent and intelligent treatment is applied right at the onset of the disease, I have demonstrated that many lives can be saved.

The fact that the invading organism is a protozoon instead of a bacterium is perhaps the reason no specific treatment has been evolved.

The first indication of Texas fever is thermic. Many hours may elapse after the first rise in temperature is noticed before the other characteristic symptoms appear. Hence, the necessity for a systematic thermic reading so that the first indication of fever may be detected and the fight begun before it is too late. The invading organism attacks the red corpuscles in the blood, breaking them down and throwing extra work on the organs of elimination in their effort to throw off the waste. The breaking down of the corpuscles is so rapid that often the liver is clogged up entirely. The liver, spleen and kidneys come in for their share of this extra work, and it is along the line of aiding these organs and also in helping to restore the red corpuscles that our line of attack must be made if we are to accomplish anything until such time as someone shall discover a method of destroying the invading organism, when Texas fever no longer will be dreaded.

When once the diagnosis of Texas fever is established, the herd should be handled so that thermic readings can be made not less than twice a day, and better three times. Take the temperature of every member of the herd every time. When once the fever is in the herd, a thermic rise to 103° F. or over should be taken as indicative and treatment begun at once.

Administer the following:

Aloes	gr. 360
Calomel	gr. 60
Nux vomica	gr. 60
Epsom salt	lb. 1

Give at one dose. This stimulates the liver to thorough action and cleans out the intestinal tract. Do not give the client any more encouragement than you would ordinarily, but tell him that if his cows are worth saving you have a fighting chance and that if you fight it right from the start you will undoubtedly save many that would otherwise die.

Start at once giving the following tonic:

℞

Reduced iron	lb. 1
Powdered gentian	lb. 2
Powdered ginger	lb. 1
Potassium nitrate	lb. 2
Powdered aloes	lb. 1

M.

Sig: Two drams twice daily.

Also in connection with this tonic, give triple arsenates with nuclein in full doses. This will be a supportive as well as tonic, and together these remedies will help to keep up the strength and build up the red corpuscles which are being rapidly destroyed.

When you have done this, you have made a nice start. Yet, the success of the treatment will be nil unless the follow-up system is good. In Texas fever there is no appetite, no thirst, no peristalsis, no normal bowel action and no milk flow in cows that were milking heavily before the onset of the disease. Right here is a good place to say that cows recently fresh and milking heavily are pretty apt to "go west" in spite of anything. Watch the bowels. Be sure they move and move freely. Treat the symptoms such as the lack of peristalsis by giving barium chlorid, 30 grains, and tartar emetic, 30 grains. I know of nothing better to start peristalsis. Until peristalsis is started, there will be no appetite, no rumination and no normal bowel action. Give this remedy three times daily. If this and the tonic do not keep the bowels open and moving properly, give the following:

Aloin	gr. 180
Calomel	gr. 60
Podophyllin	gr. 30
Nux vomica	gr. 60

If this seems too much, give less, or if not enough, give more. Always give a physic that acts on the liver and secretions. If left alone, the bowels will get very hard, and when this happens the subjects get worse rapidly. I believe this to be one of the most essential factors in the treatment of the disease. Again I say, keep the bowels open and moving freely. Cows passing "red water" or bloody urine are usually considered to be rapidly approaching death. I have saved many. Don't give up until the patient is dead. I have saved animals that have passed "red water" and were so emaciated and weak that they could hardly stand. When they begin to get weak, which they will do in spite of the supportive treatment and tonic, I commence giving milk in a drench, two or three gallons a day. Use camphorated oil if necessary. Fight to the last ditch, and you will be surprised to see how many come through alive and eventually get well. Instead of 85 per cent to 95 per cent loss there will be only 20 per cent to 25 per cent and perhaps less.

Another important step is to so handle the sick animals that not an ounce of extra energy is expended. In other words, keep them confined within a very restricted area so they will use up very little energy in moving about. Don't let them go away to pasture. If you do, many will be stricken while out and not be found until dead.

I do not claim to have discovered any treatment that is infallible or anywhere near a specific. In fact, the physic of aloes, calomel, etc., is not my discovery. All I have discovered is that if you fight from start to finish along the lines outlined, you will save many more lives than is usually expected in an outbreak of Texas fever.

I believe it is the duty of every veterinarian to strongly advise against bringing tick-free cattle into an infested territory. I am giving such advice right along. When the voters in tick-infested areas learn that only when they eradicate the tick will they be able to fully develop dairying and the livestock industry in their communities, then perhaps they will see to it that the tick goes. I am as much a booster for my locality as anyone, but I am not in favor of trying to build up the cattle industry at the expense of misinformed cattle owners

from the North and other tick-free localities. Some day, and I believe it is not far distant, the necessity will be found for eradicating the tick from every corner of the earth. Then Texas fever will no longer be known, and cattle can move freely from one locality to another.

Summary

1. Be diligent and take thermic readings every twelve hours at least. In an infected herd, a thermic rise to 103° F. or more should be considered indicative of disease and treatment started at once. The first indication of the disease is always thermic. No other abnormal symptoms may appear for some hours after the rise in temperature. Hence, the importance of thermic readings.

2. Give the dose of physic as outlined and begin at once to give the supportives and tonic. Treat the symptoms. Use barium chlorid and tartar emetic for the suspended peristalsis. Keep the bowels moving at all times from first to last.

3. Believe firmly that "while there is life there is hope" and never give up till you find the patients dead or so nearly dead that there can be no hope. Then give 10 grains of strychnin subcutaneously or intravenously and end their sufferings. Give large quantities of milk to those getting weak. Use camphorated oil if indicated. Keep everlastingly fighting. Your reward will be gratifying both to yourself and to your client.

Cystic Ovaries

J. F. DEVINE, Goshen, N. Y.

INQUIRIES WITH REFERENCE to the treatment of cystic ovaries are reaching my desk so regularly that I shall attempt to frame an article with the hope it will be at least a guide to my colleagues, who are evidently becoming more and more interested in the practical treatment of sterility.

To me the character of the inquiries that are being received now as compared with those of three or four years ago indicates that the rural practitioner is beginning to see light. If there ever was a phrase worked to death it is the one "expressing of the corpus luteum." All kinds of language has been woven around this misnomer, which to me is a strong indication that a good deal of literature has been "over the head" of most of us. I frankly confess that much of the literature accessible is indeed too much for me, "fearfully and woefully made."

I am writing this article with the hope of being of some service to the practitioner and of course know it has no place in ultra-scientific literature.

There are two important things to get clear.

First, to divide cysts into at least two classes that are plainly diagnosable. Second, the treatment. I have on my desk two inquiries that are quite characteristic.

One describes a cow suffering with nymphomania, high tail setting, falling in of the sacro-sciatic ligament, persistent bulling, all of this latter being complicated with arthritis of the hip and shoulder joints. Examination showed the ovaries to be as large as a goose egg, which proved to be cysts easily ruptured on pressure.

The other case I will quote verbatim: "A fine purebred Jersey, eight years old and an R. O. P. animal, has not been in estrum for nine months and calved some eighteen or twenty months ago. Per rectal and vaginal examination I found the left ovary was about half the size of a hen's egg presenting a rubbery firmness. Right ovary slightly larger and more firm."

The first is what we shall style in this

article peripheral cysts, the second cystic degenerated ovaries. Before taking up the handling of these cases let us try and define cystic ovaries.

A cystic ovary may be defined as one where the graafian follicle comes to the surface, and instead of rupturing as it does in physiological ovulation, fails to rupture for some reason not yet definitely understood; apparently the tunica albuginea does not become thin, allowing ready rupture, as is the case in natural ovulation, but rather stretches, resulting in an increased accumulation of fluid and giving rise to a blister-like mass varying in size from a pea to a baseball and in some cases even greater, becoming enormous and approximating a football. This cystic mass may be either single or multiple.

Ordinarily this form of peripheral cysts is comparatively benign and usually such ovaries respond readily to treatment by simply crushing the cysts and massaging the organs perhaps two or three times. This is the character of cysts that are so often found in young purebred animals that have been particularly well fed and older animals that are subjected to high protein feeding, irregular breeding and injudicious confinement. When the cysts are crushed it is usually found that the ovary of a cow, which is normally about the size in length and breadth of a lima bean but much thicker, seems to have been atrophied by the pressure of the cyst. The ovarian tissue does not give the feel of being much greater than the loose tissue that surrounds the cyst, feeling as if the ovarian tissue proper has been destroyed, but everything else being approximately normal; these are usually very satisfactory cases to treat.

The tissue seems to proliferate rapidly and normal functioning of the ovary in most cases soon becomes established. Since these cases are so often associated with nymphomania, we will discuss their handling under this heading, but would like to draw the reader's attention to the probability of this form of ovarian cyst being associated with a disturbed circulation of the ovaries and being entirely in harmony with what appears to be the predominating theory among recognized gynecologists as to the phenomena of cystic ovaries in the human family.

Formerly it was believed that rupture of the follicle was brought about by the increased tension resulting from the rapid formation of the liquor folliculi, which according to Nagel, was markedly accentuated by the pressure exerted by the luteum cells developing about its periphery. Clark, however, has shown that rupture of the follicle is a complex process and is due primarily to circulatory changes. As the period of ovulation approaches the ovary becomes engorged with blood, and the intra-ovarian tension being markedly increased, the growing ovum is forced to the surface; at the same time the circulation in the most distended portion of the wall of the follicle is interfered with, whence results necrosis at the point designated as the stigma, which eventually gives way.

If this theory is correct it would seem to explain the simple cystic ovary and how crushing the cyst and massaging the ovary usually so promptly corrects the condition by influencing the circulation.

Cystic Degeneration

Etiology — Notwithstanding that in some cases it seems quite clear, while in others it is entirely obscure, there seems to be little doubt that cystic degeneration of the ovaries is in most cases the outcome of previous infection.

It is stated that in the human family degeneration of the ovaries not infrequently occurs after acute attacks of infectious diseases such as smallpox, typhoid, scarlet fever and severe intoxications such as are caused by phosphorus, arsenic, etc. Surely those devoting special attention to sterility work in cattle must feel that by far the largest majority of the serious pathological conditions met with in the ovaries are occasioned by infection.

We are of the opinion that the custom

now in vogue of handling dairy cattle with special reference to making records has much to do with the simpler conditions that are causing temporary sterility in our fine herds, but we question if there are any or many cases of cystic degeneration of the ovaries that are not occasioned by infection.

For our purpose we will define cystic degeneration of the ovaries as differentiated from cystic ovaries as a condition in which the tissue, particularly the interstitial tissue, is thicker and the ovary is apt to be either rubberlike or doughy, with a tendency to lead into oophoritis, becoming hard and fibrous.

In the case of cystic degeneration of the ovaries most of the cysts are apt to be deep seated and if crushed one can sometimes place the end of the thumb or finger into the cavity. This cavity may be just as marked if the ovary is examined again in two or three days, showing a lack of normal circulation and regeneration, indicating as might be expected a degeneration resulting from interference with the circulation.

Ordinarily these are not the types of cystic ovaries that are associated with nymphomania, but rather are associated with the cases in which there is an absence of estrum or sexual excitement; or the estrual period is so mild as not to be noticeable even when the cow is turned into the yard with other cattle during the day. These cases are styled dumb estrum. We have seen them mingle with other animals unnoticed, but upon examination found the generative organs showing evidence of estrum, this evidence being substantiated by the cow standing for service, but her estrum being too mild to be casually observed.

This form of cystic ovaries may be associated with a cervicitis of more or less severity, or a catarrh or pyometra of the uterus. Degenerated corpus luteum is rather a common complication.

Treatment

It is our judgment that constant faithful attention to the ovaries is the remedy for these cases, frequent massaging, dislodging the corpus luteum if present, continuing manipulation of the ovaries and horns until cysts cease to form and the animal has a normal estrual period. Of course, draining the uterus if it requires it and keeping the cervix clean by frequent vaginal douching are obviously essential; however, there is much satisfaction in the knowledge that the effort will usually bring results in very obstinate cases.

There is no question but that recovery of the accompanying cervicitis is hastened by douching with a good alkaline astringent preparation, having the water hot enough to actually stimulate the parts—112° Fahrenheit is a satisfactory temperature. Massaging of the ovaries and horn should be reasonably regular, at least about once a week, and unless the case can have this regular attention for a period of three or six months if necessary then a veterinarian should not attempt to treat it. Of course it goes without saying if the animal is any distance from one's office then such handling is out of the question. Our custom is to have all such cases brought to our hospital. The use of a mixed metritis bacterin sometimes shows phenomenal results and in other cases seems to be of questionable value.

Treatment of Peripheral Cysts

If these cases are of long standing they must be treated frequently and regularly. To insure results such cases must be massaged every four or five days. If allowed to go ten days or two weeks it is just about as well not to touch them. On the other hand, if the ovaries are suffering solely with peripheral cysts and the animal is not too old, these cases many times respond so promptly to treatment as to be almost unbelievable.

I here give the history of a typical case of nymphomania: A purebred Guernsey cow, eight years old, two official records, one 700 lbs. and the other nearly 900 lbs., had not calved in three years, showed all the symptoms of advanced malnutrition, had bulled so persistently that she was

dangerous to the attendants. Upon examination the right ovary was found to be about the size of a baseball, multiple peripheral cysts which ruptured under very little pressure leaving the ovary a mass of skin-like rather than firm tissue. Left ovary not quite so large, multiple peripheral cysts, did not clean up under pressure so well. Feeding of a mineral mixture with her ration. Local veterinarian applied massage again in four days; seventh day after the first treatment cow showed no heat. Local veterinarian massaged again on the fifth day, massaging repeated again six days later. I examined the case again twenty-one days after first examination. Right ovary apparently normal, left ovary still slightly enlarged and rubber-like. Animal quiet —apparently doing well. Was in heat eighteen days later, not served, estrum seemed normal; in heat again in twenty-one days, served, conceived and calved at full term.

Milk Fever*

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Milk fever is so well known in its clinical manifestations and in its response to modern treatment that a systematic discussion of these familiar aspects of the subject would be profitless, and I propose to confine myself chiefly to a consideration of the etiology, touching casually on such other points as may have a bearing upon it.

Milk fever seems to have been recognized and recorded since the early part of the nineteenth century; in Britain, Price (1806) and Skellet (1807) being among the first to describe the disease.

That so serious an affection was not mentioned by the older writers has been explained by assuming that it only developed in such incident as justified its record towards the end of the eighteenth century and, as has been pointed out, this corresponds with the development of a more intensive system of milk production. The disease appears to have steadily increased in its rate of occurrence and was associated with a very heavy mortality.

To-day, we are happy in the possession of a specific remedy, and in the enjoyment of our possession we are apt to regard as of no account the old methods of treatment, several of which, while they met with comparatively little success, nevertheless seem to have proved of benefit in a proportion of cases, and since the matter may have some bearing on the pathology of the disease we may give it passing consideration.

I would remind you that a proportion of cases recover spontaneously and for that reason it is probable that the value of this or that treatment was frequently wrongly assessed; I shall therefore mention only a few of the more distinctive methods.

In 1806, Price advised the promotion of sweating by the use of hot packs and clothing. The procedure appears to have had little support until 1883, when Cox strongly recommended its practice, and in the following year Russell recorded that excellent results had been obtained by this treatment. In eight cases which recovered five were in extremis.

Zander, in 1874, advised the somewhat heroic practice of scarification of the croup and loins, followed by the repeated application of hot fomentations to these parts. He claimed 50 per cent. of recoveries; but the number of his cases is not stated.

Phlebotomy was long regarded as of pronounced value, both as a preventive and curative. Professor William Williams was a strong advocate of free bleeding,

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and remarked upon the improvement in the pulse which invariably followed the removal of several quarts of blood in the comatose stage. A low blood-pressure is still believed to be associated with the comatose stage, and if this be true, the beneficial effects which followed free bleeding are of distinct interest.

Chloral hydrate, first advised, I think, by McConnell, was very largely used and was claimed by many practitioners as a virtual specific, but, while it seemed to bring about a ready response in some cases, in others it appeared to have no effect. Hoare remarks, "The manner in which an hypnotic when given to a comatose subject brings about recovery is a mystery."

The year 1897 marks an epoch in the history of milk fever, as in that year Schmidt, acting on the theory that there was being produced in the udder a toxin or enzyme which brought about decomposition of the mammary epithelium, injected an antiseptic in the form of a 1 per cent. aqueous solution of potassium iodid: the results were startling in their almost invariable success.

The correctness of Schmidt's theory was later disproved in that it was shown that what he had regarded as evidence of epithelial disintegration were normal tissue elements, and it was thereupon suggested that the iodin which was injected probably possessed some specific power in neutralizing the pathogenic factor.

It should be noted (and this fact has hitherto been largely overlooked by myself among others) that a comparatively small quantity of the solution was at first injected; a quantity, in fact, even less than the quantity of milk removed from the udder previous to injection. Schmidt did not attempt to effect mammary distension, but was content to introduce a small quantity of solution which he then proceeded to "massage throughout the gland."

Schmidt records that some practitioners employed injections of small quantities of a concentrated solution of potassium iodid (10 grams in 40 grams of water), and that in several cases recoveries were obtained. Peter injected intravenously 10 grams of potassium iodid in two liters of water with success; Wessel claimed 75 per cent. of recoveries with this treatment, the value of which is vouched for by other writers, including Hauptmann and Schmidt himself.

I have perhaps sufficiently indicated my point that these obsolete procedures should not be forgotten in that they may have some bearing on the pathology of the disease.

At first, practitioners were enjoined to be careful to exclude the entrance of air during the injection, but this was a matter of some difficulty and it was found that when air was accidentally injected with the fluid, no harm resulted: it was later believed to be of actual value in aiding the dispersal of the fluid throughout the gland and for this reason it was eventually adopted as a routine method; the udder was first partially injected with the iodid solution and thereafter air was pumped in.

Anderson, of Skanderborg, was the first to practise inflation with air alone, and the older methods were soon replaced by simple mammary insufflation and distension.

So it came about that the veterinary profession was privileged to have presented for its solution a problem in etiology as intriguing, surely, as any which has confronted medical science.

An apparently healthy, prime milch-cow, after performing successfully and without mishap a perfectly normal function i. e., parturition, is suddenly struck down by a serious disease, all cases of which present similar characters of occurrence and the same general train of symptoms: a disease from which, in all probability, she will die unless she be subjected to a mechanical process almost ridiculous in its simplicity ; whereupon she makes a recovery that is not only complete, but almost as rapid as was the attack. The problem is the more attrac-

tive by reason of its apparent simplicity.

We seem to be confronted with an enigma-tantalus: could we comprehend the nature of the disease, the meaning of the dramatic recovery following inflation should become evident; could we understand the action of the remedy, the nature not only of milk fever, but of the whole group of eclampsias to which it appears to belong, should become capable of conception.

Conditions of Occurrence

In discussing the conditions of occurrence, I propose to take a course which is open to your criticism. I shall assume that the disease we call "milk fever" occurs, not only in the parturient cow, but at any stage of lactation and even in the non-lactating cow; that it need not manifest itself in the form so familiar to us when it occurs during the puerperium, but may appear some few weeks after calving as a sub-acute dyspepsia with or without attendant convulsions ("post-parturient dyspepsia"); that it also occurs in the parturient goat, ewe, and sow, and in the goat and ewe during late lactation.

I do not scruple to make these suggestions, because I believe that in them I am on fairly secure ground, and I would further suggest, although I am now in the realms of speculation, that some cases presenting the milk fever syndrome which occur in the fed bullock (so-called "staggers") are allied in their essential pathology to the disease as it occurs in the parturient cow.

My arguments that all these conditions are of the same essential pathology are simple:—

(1) All respond readily to simple mammary insufflation (with the exception of course, of "staggers" in the bullock).

Now, since mammary insufflation represents a simple mechanical process—(the results are the same, whether air, antiseptic solutions, or sterile water be injected)—then it seems reasonable to believe that whatever effect is elicited, it must be essentially the same in all cases and that the diseases which readily respond to the production of that effect must be the same in their essential pathology.

(2) The factors, other than parturition, which appear to predispose to the occurrence of these conditions are similar to those of milk fever.

(3) All, with one exception, present clinical symptoms apparently identical with those of milk fever, but even the exception, post-parturient dyspepsia, occasionally terminates with symptoms identical with those of milk fever.

Attempts have been made in the past to differentiate several types of parturient milk fever. While that may be convenient for the purpose of clinical reference, I believe it is wholly wrong when considering the disease in its general pathology. I believe that, in general, all cases of milk fever in the parturient cow can be regarded as exhibiting a well-defined syndrome occurring in two stages:—(1) tetany and convulsions succeeded by (2) coma. In some cases the first stage is pronounced and lengthy, in others the second stage develops rapidly. To recognize several clinical types is fallacious and tends to confuse the real issue.

Now, if I am right (I say if I am right) in assuming that these diseases represent one pathological entity, then let us see what inferences can be drawn from this premise.

(a) Milk fever occurs in the lactating cow, long past her puerperium; then parturition is not an essential factor in the etiology.

(b) Milk fever occurs in the non-lactating cow in whose udder milk secretion has entirely ceased; therefore lactation is not an essential factor in the etiology.

On the other hand, since the disease is so commonly associated with the first milk-flow following parturition both these factors must be regarded as very important predisposing causes.

Let us now turn to a consideration of the factors which appear to determine the occurrence of the disease as it is met with in its most familiar form, i. e., following

parturition in the cow. It is to be realized that these factors are predisposing and not exciting causes. There are many cases of milk fever in which some of these factors are absent, while on the other hand several are associated with parturitions that are not followed by milk fever.

Predisposing Causal Factors

(1) Distribution.—The disease occurs most commonly in certain districts, and practitioners have frequently noted that individual farms furnish cases of milk fever, whereas the disease is seldom encountered in others. Again, as many as five or six cases may occur at short intervals in a herd in which previously the affection was rarely seen. This might indicate the presence of a factor to which all the animals are exposed and to which certain are susceptible or, conversely, the absence of some factor (dietetic or otherwise) the presence of which would tend to prevent an attack. It has been considered that such a factor might be calcium, and the point will be referred to later.

(2) Age.—Cows are most commonly attacked between the fifth and the tenth years of age. It has been noted that the commencement of the period of greatest susceptibility corresponds with the period of greatest milk yield. Primiparæ are very rarely attacked.

(3) Type of Cow and Conditions of Keep.—While deep-milking cows in high condition are most liable to attack, milk fever is occasionally encountered in poor, lean animals; but more especially when these have been the subjects of generous feeding towards the end of pregnancy. The danger of a rich nitrogenous diet in the last stage of pregnancy has long been recognized, but cases occur in cows at pasture, especially on lush, rank grass, and a condition of sub-acute dyspepsia is not uncommonly noted previous to and succeeding the attack. Great difference of opinion is expressed as to the conditions of keep most likely to produce the disease. While some have observed the majority of their cases in closely confined stalled cows, others have experienced a greater number of cases when the cows are out at grass in the summer.

(4) Conditions at Parturition.—In practically every case the birth has been easy and uncomplicated. The disease may follow abortion, and the retention of the placenta is a fairly common concomitant. It has been observed that milk fever seldom, if ever, follows the birth of twins, but the point wants confirmation. It has also been observed, but again confirmation is lacking, that the period of non-lactation previous to the calving is usually short.

(5) Weather.—Attempts have been made to associate the attack with various types of weather, and it has been noted that periods of hot, muggy, thundery weather are likely to produce a crop of cases.

(6) Fatigue and Excitement.—Forced exertion, fatigue and excitement must be regarded as predisposing causes. The common occurrence of milk fever in freshly-calved cows which have been sent a long journey by rail or road has often been remarked.

(7) Influence of Milking.—The complete emptying of the udder after calving is an important causal factor. While in many cases the milk secretion is markedly diminished prior to the appearance of the notable symptoms, the sudden and complete withdrawal of milk may precipitate the onset of the attack. On the other hand, the avoidance of complete milking for the first 72 hours after calving is perhaps the most valuable means we possess for the prevention of the disease. Cows suckling their calves are rarely attacked; this is usually explained on the ground that the milk is drawn off in small quantities, and that the excitement consequent upon separating the calf from the cow does not occur.

(8) Liability to Recurrence.—That the disease shows a tendency to recur with each parturition is well known. This does not necessarily mean that one attack renders the subject more susceptible to another; it rather suggests that some

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factor which ordinarily plays an important part in the resistance of the body to the disease is absent.

Let us now consider briefly those other conditions which respond to mammary inflation, and which I believe to be very closely related to parturient milk fever.

A.—Post-Parturient Dyspepsia

This condition is frequently met with in the South and West of Scotland, but appears to be more common in some districts than in others. It most frequently affects heavy milking cows in high condition. While cows which have reached their prime are principally affected, it, more commonly than milk fever, attacks young animals and even heifers. The influence of rich nitrogenous feeding is well recognized, and the disease is almost invariably associated with an easy parturition. A subject of post-parturient dyspepsia is likely to be similarly attacked after the next calving, or she may then develop typical milk fever.

It will be seen from the foregoing that the predisposing factors are very similar to those of milk fever.

The attack usually develops within two to four weeks after calving. The symptoms are somewhat indefinite and, in general, represent a sub-acute dyspepsia. Dullness is followed by suspension of appetite and rumination; the intestinal movements are sluggish, the feces being rather solid, dark colored and containing excessive mucous film; the milk secretion is markedly diminished and the udder is distinctly flaccid. The skin becomes dry and harsh, and the coat uneven and dirty in appearance. There is no fever or other marked constitutional disturbance, but occasionally there is developed a persistent and penetrating mawkish odor which is principally exhaled from the mouth, but which also affects the milk.

These symptoms usually persist for a period varying from one to three weeks, when they may clear up spontaneously, although recovery is usually hastened by aperient and stomachic treatment. Insufflation of the udder, in the great majority of cases, brings about a ready response, the symptoms quickly disappearing.

It will be noted that no tetanic or convulsive symptoms have been described, but, in a proportion of cases the symptoms of dyspepsia are succeeded by those of acute milk fever.

B—Milk Fever Complex Occurring at Periods Other Than the Puerperium and Responding to Mammary Inflation

Disease conditions which cannot be clinically distinguished from milk fever and which are often referred to as "staggers" present themselves at every period of lactation. In many of these cases the cause of the attack is obscure, but three factors may be mentioned:—

(1) Dietetic Error.—Under this may be included overfeeding or the use of a too-highly nitrogenous diet. Cases are also met with in cows grazed on rich pastures, especially those carrying a high proportion of clover. These conditions, which are sometimes referred to as "stomach staggers," also occur in sheep and goats, in which animals, also, inflation occasions a rapid recovery.

It is worthy of notice in this connection that digestive disturbance is frequently also associated with parturient milk fever, and is the characteristic symptom in post-parturient dyspepsia.

The term "staggers" is applied without distinction to a number of conditions of obscure etiology. Many cases do not respond to inflation, and it has been observed that these differ clinically from the milk fever complex in exhibiting diarrhea, rather than constipation, but the point requires to be verified.

(2) Excitement and Fatigue.—While, as has been already mentioned, these may act as important causal factors in parturient milk fever, they are also known to be the means of precipitating the syndrome as it occurs at a period other than the puerperium, not only in the lactating, but in the "dry" cow and ewe. There are several records of the occurence of such

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cases at markets and fairs. Inflation is usually followed by rapid recovery.

(3) Estrum. That the milk fever syndrome in the lactating but non-pregnant cow is frequently associated with the period of estrum, especially the first period following parturition, has been recognized by several practitioners. Since considerable excitement may obtain during the estral period it is possible that in this we have the cause. On the other hand, the part played by endocrine action in the production of the estral cycle may yet be found to furnish the explanation.

The findings at autopsy have been meager and indefinite. Among the more significant conditions described are—a certain degree of engorgement of the abdominal vessels, and sub-involution of the uterus. This latter condition is capable of demonstration during life, but a greater consensus of opinion is required as to its relative frequency as compared with the normal.

Several writers, including Porcher, Albrecht and Nocard have found albuminuria during the course of the attack, but its presence is inconstant; this does not exclude the possibility of nephritis, as it has been shown by Browne (1926) that nephritis need not necessarily be associated with an albuminuria. One would, however, expect to find in such a condition an appreciable rise in the urea and uric acid of the blood, but, according to Hayden and Sholl (1926), the average increase in these nitrogenous end-products in milk fever does not seem to indicate any considerable impairment of renal function. Nevertheless, a series of careful histological examinations of the kidneys seems desirable.

Theories Upon the Etiology of Milk Fever

Cerebral Anemia.—It has been postulated by several writers that milk fever represents a cerebral anemia consequent upon an assumed vaso-motor failure and the resultant collection of large quantities of blood in the abdominal area; the decreased abdominal pressure following parturition and the mammary hyperemia accompanying the commencement of lactation have been advanced as contributory causes.

In support of this hypothesis, several cases have been recorded in which the appearance of the comatose stage of milk fever has been observed following postpartum hemorrhage, but all attempts to reproduce the typical symptom-complex by copious bleedings have failed. Moreover, as has been pointed out by Marek, in such cases as have been recorded as simulating milk fever, not only blood, but extraneous matter had gained entrance to the peritoneal cavity. In one such case, in which coma was complete, Gebauer mistook the condition for milk fever and inflated the udder; twenty minutes later the cow showed signs of returning consciousness which became complete and continued for about an hour, when coma returned. In other cases of intra-peritoneal hemorrhage inflation has produced no appreciable effect.

The theory further postulated that inflation, by increasing mammary pressure, drives the blood out of the udder into the general circulation, or in some manner causes contraction of the abdominal vessels and so overcomes the cerebral anemia. While one can no longer regard cerebral anemia as the primary factor in the pathogenesis of milk fever, it is conceivable that a failure of the vaso-motor center may be present in the comatose stage; the theory, however, does not attempt to explain the process of its causation. That splanchnic (sympathetic) stimulation may follow inflation seems probable, and will be discussed later, but the increase in the general blood pressure due primarily to udder distension and occlusion of the mammary vessels would be so slight as to be almost negligible.

Cerebral Congestion.—The earlier theories of Franck and Professor Barlow, of Edinburgh, that the disease depended upon sudden cerebral congestion are now regarded as untenable.

Organic Lesions.—The older hypotheses postulating such conditions as thrombosis, fat embolism and apoplexy

fell in face of the rapid and complete recoveries which follow mammary insufflation.

Bacterial Intoxication. — In 1885, Schmidt-Mülheim, in advancing the theory that milk fever represented a form of botulism, based his argument upon certain resemblances in the clinical symptoms. This idea has recently been brought forward again by Pugh (1924), who suggests that the disease results from the absorption of botulinus-like toxins to which the animal is predisposed by the presence of a ketosis which, in turn, is consequent upon faulty fat metabolism at parturition.

This theory has already been rather fully criticized by Professor Henry Dryerre and myself (1925), but attention may here be drawn to the following points:—

Little and Wright (1925), in a quantitative estimation of the acetone bodies in the blood and urine of cases of milk fever, found that there was no significant increase in these bodies as compared with the normal cow, i.e., that there is no ketosis. Hayden and Sholl (1925), in an examination of the urine in twelve cases of milk fever, concluded that acetone bodies "do not seem to be present in consistent pathological quantities." In six of their samples, however, the urine was acid in reaction so that while the possibility of an acidosis is not wholly excluded there appears no justification for the theory that milk fever is caused by a botulin-intoxication consequent upon ketosis.

If milk fever be due to the action of a bacterial toxin, then one must believe that the mechanical process of mammary inflation will not only immediately overcome the effects of that portion of the toxin which has already entered into combination with the body tissues, but will destroy the toxicogenic property of the bacterium itself.

Such conclusions cannot be accepted.

Several writers have regarded the disease as an intoxication of bacterial origin and have suspected the uterus, vagina and mammary glands as the probable sites of infection. It has been thought that toxins might be developed in the uterus during the decomposition of the lochia, but these have never been identified as causal factors, and no micro-organism has been found experimentally to produce the disease.

But, while, as has been indicated, I believe that the theory of bacterical intoxication is untenable, the idea of an auto-intoxication is much more attractive.

Auto-Intoxication consequent upon Anaphylaxis.—Marek suggested the possibility of an auto-intoxication following anaphylaxis produced by the absorption of specific proteins from the placenta or the glandular cells of the mamma, but attempts to reproduce the phenomena of attack by intravenous and intraperitoneal injections of placental emulsions, milk and colostrum proved negative.

Hynd (1925) has recently investigated by polariscope tests the nature of the urinary protein in eclampsia of woman; his results appear to show that lact-albumin, as distinct from serum-albumin, is present. This suggests that lact-albumin is re-absorbed into the blood in which it must be regarded as a foreign protein. Hynd says "possibly it may be that small amounts of lact-albumin may find their way into the blood-stream, but that during lactation as a rule immunity to this foreign protein is established. When this immunity is not established, or when a sudden release of lact-albumin occurs corresponding to a shock dose of foreign protein, then eclampsia is the result."

It is possible that this may also apply in milk fever, and further investigation along these lines is highly desirable.

Auto-intoxication consequent upon Endocrine Disorder.—In 1925, my colleague, Professor Henry Dryerre, and I formulated a hypothesis as to the nature of milk fever and those diseases which resemble it in their clinical symptoms and in their response to mammary insufflation. We were of opinion that some light could be thrown on the problems by a considera-

tion of the disease as representing an endocrine dysfunction.

A detailed discussion is unnecessary here, and it will suffice to briefly outline the theory.

I would remind you that the para-thyroid glands are generally assumed to play a part in the neutralization of toxic metabolites, and it has been shown that they perform the important function of regulating the calcium content of the blood. While the mechanism of their action is by no means clear, it is known that removal or atrophy of the para-thyroids gives rise to a condition of tetany.

It has been found that, in conditions of para-thyreopriva there results a deficiency of calcium in the blood, and since the injection of calcium temporarily relieved the symptoms, it was concluded that the tetany was due to a calcium deficiency caused by the para-thyroidectomy. Later, the presence of a substance, methyl-guanidin was discovered in the urine of dogs, deprived of their para-thyroids and Paton and his co-workers found that injections of guanidin into the muscles of mammals produced symptoms exactly similar to those occurring during para-thyreopriva.

In animals in which deficient functioning of the para-thyroids has been induced, in addition to the symptoms of tetany followed by coma, there also occur dilation of the pupil, tachycardia and a depressed intestinal movement.

In cases of para-thyroid hypofunction the animal is able to meet the usual metabolic requirements of the body, but it possesses a lessened ability to combat the effects of calls made upon it in excess of the normal. Such a condition is known as latent tetany, and we suggest that cows exhibiting milk fever and the allied diseases to which I have referred may be suffering from this defect. The animal during pregnancy is able to meet the strain of increased metabolism involved, but is on the border-line; following parturition there is a great increase in protein metabolism, and in the necessity for an increased destruction of toxic metabolites. The more intensive the feeding, the greater will be the danger, for it has been shown that a diet rich in proteins predisposes to the formation of guanidin compounds. At this period, too, the blood calcium, already deficient in quantity, undergoes heavy loss by reason of the secretion of colostrum, and heavy calls are made upon the para-thyroids for its replacement and stabilization in the tissues. Hence the para-thyroids are unable to cope with the demands made upon them; toxic metabolites gather in the blood with their consequent sequelæ.

The typical signs of guanidin poisoning are manifest, not only in milk fever, but in the nervous complications of post-parturient dyspepsia and in those cases of "staggers" which respond to inflation.

The circulation of adrenalin in the blood-stream is known to play an important role in controlling the rate of oxidation in the tissues, and there is reason to believe that in the conditions under consideration there also exists a sub-normal or only normal secretion of adrenalin, so that the increasing toxemia fails to be counteracted by an increased outpouring of adrenalin.

In cases of dystocia we may believe that the adrenals are profoundly activated in response to the afferent stimuli produced by severe labor, and in this way there will occur a profuse outpouring of adrenalin into the circulation to be followed by a rapid oxidation of the excessive toxic metabolites in the blood and the consequent prevention of the symptoms of milk fever.

In this way, too, we would explain the curative effects of mammary insufflation. We suggest that the inflation gives rise to powerful afferent stimuli which reflexly produce the outpouring of adrenalin; oxidation is hastened, the toxins are burnt up and the blood pressure raised.

The distension of the udder by inflation will, by retarding the formation of milk, conserve the supply of calcium in the blood. This may also explain the efficacy of a restricted withdrawal of milk in the

prevention of the disease. It has further been shown that small doses of adrenalin increase the excitability of muscle and that the presence of guanidin increases enormously sensitizing effects of adrenalin upon muscle, while the administration of a small dose of adrenalin to an animal with its para-thyroids removed will precipitate the onset of tetany.

From these observations we can correlate the fact that fright, worry or excitement may usher in an attack of the milk fever syndrome, both as it occurs in the lactating. but non-parturient cow, ewe and goat, and in the non-lactating cow. The stimulus of fright or of the distress occasioned by restraint, etc., is sufficient to cause only a comparatively small secretion of adrenalin—enough to sensitize the tissues, but not enough to hasten oxidation rapidly. Likewise an easy birth, by its relatively small disturbance to the animal, elicits only a small secretion of adrenalin.

We may believe that guanidin, which is a by-product in protein metabolism, is normally present in the blood (but especially at parturition) in greater or lesser amounts; but in parturient milk fever and the other allied conditions it oversteps the danger threshold. As has already been noted, the undoubted beneficial effects which followed free bleeding were difficult of explanation, but if milk fever represents, in part at least, an auto-intoxication by such substances as guanidin, the value of phlebotomy can be understood as representing a removal of toxin from the blood. In the same way, the preventive value of aperients in the last stage of pregnancy is explained on the ground that the formation of guanidin in the bowel and its absorption are facilitated by intestinal stasis. Again, since guanidin production is more readily occasioned by a highly nitrogenous diet, we can correlate the fact that a subsistence diet of straw and roots in the last week or two of pregnancy tends to prevent an attack.

The occasional failure of mammary insufflation to effect a complete cure may be traced to the fact that the insufflation was not pursued to a sufficient degree and hence the necessary stimulus for the reflex secretion of abundance of adrenalin was not initiated or, alternately, that the case represented an unusually heavy guanidin intoxication. The necessity in certain cases for a second or third insufflation of the mammæ may be readily deduced from our hypothesis; the first treatment gave a secretion of adrenalin only sufficient to carry the animal over a limited period, but not past the danger threshold. The subsequent insufflations completed the cure.

This hypothesis has received considerable support in experimental and clinical evidence, thus:—

Little and Wright (1925), in an investigation of twelve cases, found that the cows when "down" with milk fever showed an average fall of 50 per cent. in the calcium content of the blood. Further, the fall in the calcium content always ran parallel with the severity of the symptoms. After inflation the blood calcium again rose.

These workers state that benefit has been derived from both intravenous and intramuscular injections of calcium chlorid in 10 per cent. solutions; 30 to 40 c.c. being injected, and repeated if required in six to eight hours. When possible, calcium lactate can be given orally every three hours with benefit.

Little and Wright do not agree that the lowered blood calcium should be interpreted as a para-thyroid failure and suggest that it may partly be accounted for in a deficiency of lime in the rations, or by confinement in stalls during the winter when the animals are denied a sufficiency of sunlight and an adequate supply of fresh green (vitamin containing) foods which are necessary for the proper assimilation of lime. They point out that while the cow suffers very considerable loss of calcium during pregnancy, the sudden loss of calcium at the commencement of lactation is particularly striking. They estimate that the secretion of half

a gallon of colostrum would be sufficient to deplete the blood of the whole of its lime, supposing there was no reserve store of lime in the body from which the blood might be replenished.

It will be apparent that if inflation brings about an increased adrenalin outflow a rise in blood pressure should follow. Seitter, by insufflating the udder of healthy cows, found that the normal blood pressure could be raised within half an hour by an average of 55 per cent.; he also found that blood pressures lowered to 51 per cent. of normal as the result of the administration of drugs such as choral hydrate, could rapidly be restored to normal by insufflating the udder. The effects of the insufflation appear to have lasted about eight hours. Similar, but much less rapid, results were obtained by the injection of adrenalin (method and quantity not stated). If one assumes that mammary insufflation stimulates (directly or indirectly) the adrenal glands, such effects upon the blood pressure are readily comprehensible.

Again, it is well known that the production of an increased adrenalin secretion immediately raises the glucose content of the blood, and hyper-glycemia will result. Widmark and Carlens (1925), experimenting upon normal lactating cows and goats, found that mammary inflation caused an immediate hyper-glycemia of short duration (it appears to reach its maximum in about 60 to 90 minutes), and this was most pronounced in heavy milking animals. After reaching its maximum the hyper-glycemia rapidly falls.

Adrenalin has long been recommended as an ancillary to udder inflation. Again since the secretion of adrenalin brings about uterine contractions, it is interesting to note that mammary inflation produces contraction of the sub-involuted uterus; this cannot, however, be appreciated in all cases and further observations on the point are required.

While the work of Seitter and that of Widmark and Carlens would appear to support our theory that inflation causes an outpouring of adrenalin, it should be noted that both an increase in blood pressure and an increase in the blood sugar should follow excessive pituitrin secretion.

It is accepted that extracts of the posterior lobe of the pituitary and of the adrenal medulla mutually facilitate each others's action.

From what is known of the action of pituitrin one could understand its proving of benefit in milk fever, but the manner in which its secretion could be induced by inflation of the mamma is difficult of conception. It is conceivable that there is a connection between mamma and pituitary through the medium of the uterus with which both are known to have an endocrine relationship, and that inflation of the mamma by influencing the production of mammary hormone may exert some action on pituitary secretion. Nairn (1925) has suggested that the secretion of the mammary hormone is arrested by inflation and that, indirectly, the adrenal-thyroid-ovarian group is stimulated.

While this matter is all highly speculative in character, mention of it here is, I think, justified.

As has been promised Professor Dryerre and I are engaged upon research into such points in our theory as are capable of demonstration.

It is much to be regretted that unforeseen circumstances have delayed this work, the results of which are still insufficient upon which to base precise conclusions. It may, however, be said that our research is on similar lines to that of Little and Wright, with whose conclusions it appears to be in agreement.

Hypo-glycemia.—Basing their arguments on the reasonable assumption that during lactation the blood glucose is converted into the lactose of the milk, Widmark and Carlens believe that milk fever represents a hypo-glycemia. This conversion they say must be pronounced in a heavy milking cow and, as a consequence, a hypo-glycemia is occasioned during the sudden onset of lactation after

calving. They suppose that distension of the udder causes an arrestment of lactose formation and therefore of glucose absorption from the blood, so that a hyper-glycemia develops, but, "as soon as the mechanism of adjustment has begun to work through the appearance of the hyper-glycemia, the hyper-glycemia diminishes."

It is difficult to apply this hypothesis to those cases presenting the milk fever complex which occur during late lactation or non-lactation, nor can it be correlated with the occurrence of post-parturient dyspepsia and its cure by inflation. Further, if this theory be correct there seems no reason why milk fever should not occur in practically every heavy milking cow at parturition. Again, the means by which an induced hyper-glycemia of 90 minutes' duration restores the mechanism of glucose stabilization in the blood for the remainder of the period of lactation is left unexplained.

While there seem good grounds for accepting the conclusion that mammary inflation occasions an immediate, if short, hyper-glycemia, it does not follow that upon this effect its curative action depends nor does the finding necessarily imply that a hypo-glycemia is the determining factor in milk fever. Indeed, as will be shown presently, there is reason to doubt the existence of a hypo-glycemia during the attack.

Insulin the product of pancreatic endocrine tissue when given in overdosage causes a profound hypo-glycemia with symptoms which bear a resemblance to those of milk fever, and in view of this, Widmark and Carlens (1924) and Neefs (1925) advised the use of the intravenous injection of glucose (20 grams) as a curative measure.

Maguire (1926) found, in an estimate of the blood-sugar in five cases of milk fever, that hypo-glycemia existed and that recovery could be obtained by injecting glucose intravenously. He also found that inflation produced a transient increase in the blood sugar (thus further supporting the findings of Widmark and Carlens).

Maguire believes that milk fever represents an excessive activation of the endocrine tissue of the pancreas resulting in hypo-glycemia.

On the other hand, Hayden and Sholl, in a determination of the blood-sugar content in milk fever, obtained results which are, in the main, quite contrary to those of Maguire.

In an examination of forty-four normal cows they fixed an average reading of 51-75 mgms. of sugar per 100 mls. of blood. This figure they considered as probably low. The blood collected from twenty-seven cases of milk fever gave an average reading of 95-82 mgms. per 100 mls. In nine of these cases another sample of blood was taken after recovery (actual time not stated) when an average reading of 72-75 mgms. was obtained.

The averages given, are, in themselves, somewhat misleading, as very great variations were found to occur in the sugar content of blood samples collected during the attack—the figures ranging from 35-7 (hypo-glycemia) to 190-4 (pronounced hyper-glycemia).

Hayden and Sholl conclude that there is a hyper-glycemia in milk fever and the high sugar content falls after recovery upon inflation.

It is to be noted that this does not necessarily conflict with the statement of Widmark and Carlens that inflation causes a transient hyper-glycemia, as Hayden and Sholl took their second sample "after recovery," when presumably the hyper-glycemia had declined.

Little and Keith (1926) failed to demonstrate hypo-glycemia in a limited number of cases of milk fever.

It would appear from the available evidence that, while hypo-glycemia may occur during the attack, the majority of cases of milk fever show a heightened blood sugar content and that, therefore, when hypo-glycemia does occur it is incidental to the disease. In other words, if a hypo-glycemia were the determining

factor in milk fever, then one should find it present in every case.

It may be noted from the foregoing that the symptoms of milk fever are closely simulated by:—

(1) Guanidin intoxication associated with calcium deficiency.

(2) Insulin overdosage.

That there may be a connection between insulin secretion and guanidin production seems possible, for it has been demonstrated by Secker (1925) that both insulin and guanidin possess similar actions upon the permeability of cells, while Burns has shown that the production of guanidin is stimulated by insulin.

It is conceivable that the symptoms which follow insulin overdosage do not depend upon the resultant hypo-glycemia but upon the liberation of guanidin.

Several observers have suggested the possibility of an inter-relation between milk fever and parturient eclampsia of woman.

For some time past I have had the privilege of co-operating with Dr. Francis J. Browne, of Edinburgh, now Professor-elect of Obstetrics in the University of London, in considering the practicability of inflating the breasts in parturient eclampsia.

The human mamma differs from that of the cow in that it is made up of from 15 to 24 lobes. Each lobe consists of several lobules which, in turn, are composed of large masses of acini. Each lobule is provided with a duct which, meeting others, unites to form a single larger canal for each lobe. Each of these lactiferous ducts opens separately at a minute isolated orifice on the surface of the nipple. To effect distension of the breast with air, it follows that each of the lactiferous ducts must be separately inflated through its orifice.

In our early attempts we employed a miniature "teat siphon," similar in form to that used for the cow, but this was soon found to be useless, as we could not obtain a solid-pointed instrument of sufficiently fine calibre to effect our purpose. We then devised a simple cannula corresponding in calibre to a No. 25 hypodermic needle, and it was found that this could be introduced into several of the ducts without injury. By this means partial inflation of the breast has been obtained in two cases of eclampsia, but complete distension has not yet been effected.

While the riddle of milk fever still remains unsolved, we may be encouraged in the realization that within recent years real progress has been achieved. This has been largely accomplished by the experimental research to which reference has been made in this paper. Baseless theory has given place to reasoned hypothesis which, later, may be put to demonstrable proof.

Our quest is no mean one. The key to the problem may give entry to a new field of knowledge in comparative medicine.

The farms of the middle west are annually growing, training, educating and delivering from 300,000 to 400,000 young folks of working age to our cities.

The production of cheddar cheese of high quality not only requires the use of milk of good quality, but also proper equipment to facilitate manufacturing and curing.

Cactus is increasing at the rate of a million acres a year in eastern Australia. It has been reported that approximately sixty million acres of Australian land is occupied by cactus.

Texas had 1,776,000 goats on January 1, 1926, and in 1925 eight million nine hundred pounds of mohair were produced in that state.

Female Angora goats produce the maximum of mohair at two years of age. Wethers usually produce the maximum of mohair at three or four years of age.

Bovine Coccidiosis*

RONALD GWATKIN, Ontario Veterinary College

Coccidiosis in cattle in British Columbia was reported by Bruce in 1921 (Jour. A. V. M. A., March, 1921). He found a dysentery due to coccidia affecting range cattle of all ages. It occurred in the dry interior of the province and usually in the winter. Out of one hundred and sixty-five visibly affected animals fifty died. Young animals suffered the most severely. Fatal cases died usually in from two to five days after showing symptoms, and within twenty-four hours of death were subject to fits of short duration, death occurring in convulsions. The principal lesions were confined to the rectum in older animals and to the small intestine in young calves. The coccidium differed from available accounts of Eimeria zurni and also from that described by Smith and Graybill (Jour. Exp. Med., Vol. 28, No. 1, 1918). The name Eimeria canadensis was proposed for it.

Marsh (Jour. A. V. M. A., February, 1923) reported coccidiosis in Montana. It occurred principally in calves about six months old, with some cases in yearlings and two-year-olds. Older cattle were occasionally affected. All cases occurred between November and March. Mortality was estimated at twenty-five per cent.

Hutyra and Marek (Path. and Ther. of Diseases of Domestic Animals, Vol. 2) state that the disease occurs almost exclusively during the warm season between the months of June and September. It occurs principally in mountainous parts.

On August 28th we received samples of feces from G. R. Booth, B. V. Sc., of Palmerston, who informed us that he had a few cases of bloody diarrhea. The trouble had appeared first in an old cow which had apparently recovered two weeks previously. On August 23rd the condition was noticed in a yearling heifer. Three more were affected August 26th. The cattle were on alfalfa and red clover pasture. The sample contained numerous oöcysts.

*From Report of the Ontario Veterinary College, 1925.

The affected premises were visited in company with Dr. Booth. The following particulars were partly ascertained at that time and partly supplied by Dr. Booth later, to whom we are indebted for material and information.

History

The owner had thirteen head of cattle. Trouble had commenced about three weeks previously in a cow which passed some bloody feces, but was not noticed to be straining. Two weeks later two more cows developed the condition. Next day three more were affected. In all, at the time of our visit, nine animals had been noticed to have had diarrhea. They were an aged cow, three four-year-olds, two animals two years old and rising two, and three yearlings.

A bull was kept and cows had been regularly brought in from the neighbors' premises. This animal was stabled and showed no trouble. A small creek passed through the front of the farm. The cattle had access to it at times but were usually watered from a well in the barnyard.

The last cattle purchased were two animals that had been brought in with two carloads of cattle from Toronto in the fall of 1923. The man who had brought these cattle from Toronto had no trouble of any kind. One of the purchased animals was killed last winter. The other was still on the premises. Neither had been observed to have had diarrhea at any time. On the farm on the east side, from which the creek flowed, there had been an outbreak of diarrhea in the fall of 1924 which was believed at the time to be due to turnips. There had been no trouble on the other farms adjoining or on the corners.

Samples of feces were taken from some of the animals that had recovered and some that had not been affected. No coccidia were found in them.

On September 12th a sample of feces was sent in from a second herd three miles away, from a yearling with bloody diarrhea, and was found to be loaded with oöcysts. This was the only animal affected in this herd.

Later, another herd was found to be affected. It was also three or four miles from the first. In this herd two calves, six to seven months old, were the only ones that appeared to be affected. Many oöcysts were seen in the feces.

A case occurred in a six-months-old calf on the farm across the road in which there had previously been no trouble. Oöcysts were present in the feces in large numbers. This was the only case on the premises. This made the fourth herd within a short distance in which the trouble occurred.

Symptoms

Young animals showed the most marked symptoms. Straining was violent, a good deal of blood was mixed in the feces and clots of almost pure blood were passed at times. Temperature did not go above 103° F. Animals became rapidly emaciated and the coat was rough. Milk secretion rapidly diminished, but picked up as quickly when straining ceased. Appetite usually remained fair, except in some cases in which rumination ceased for about a day. Most of the affected animals did not seem much troubled even when passing a considerable amount of blood. In some cases the feces were not very liquid even though quite a large amount of blood was present.

Bruce refers to fits occurring in fatal cases, in which the animal appears frightened, staggers, may brace itself by resting the head on the ground with the forelegs widely spread, falls over and froths a little at the mouth, and may get up in a minute or two as if nothing had happened. None of the cases reported here were fatal and this condition was not observed. In one instance nervous twitching was observed in a calf, which finally recovered.

Treatment

The treatment recommended was that employed by Bruce in British Columbia, namely, sulphate of iron 2 parts, sulphur 2 parts, salt 6 parts, fed at the rate of 50 lbs. of the mixture with 100 lbs. of linseed oil-cake meal to 100 head per day. As a matter of fact most of the affected animals appeared to recover so rapidly that in some cases the treatment was not applied. Liming and ploughing of infected land was recommended where possible, as also proper disposition of feces in stabled animals.

Experimental

Four cockerels and pullets about two months old were fed 2.5 cc. of feces that had been kept for two weeks, part in the incubator at 37° C. and part at room temperature. Even after two weeks many of the oöcysts were unchanged in appearance. Two white rabbits were fed with 5.0 cc. and later 2.5 cc. each of the same material. All were kept under observation for a month. They remained normal and no coccidia were observed in the feces.

A number of oöcysts in the early granular stages were measured. They varied in size from 15 to 13 by 18 to 16 microns. Spherical forms were common and varied in diameter from about 14 to 17 microns.

Summary

Coccidiosis was diagnosed in four herds on farms within a few miles of each other. There were no fatal cases and recovery in most cases was fairly rapid. Temperature did not go above 103° F. Treatment recommended was that advocated by Bruce. Young fowl and rabbits were not affected by being fed feces containing oöcysts which had been kept for two weeks. A study has not yet been made of the parasite.

Avian coccidiosis is rather widespread and destructive to young chickens. This malady is characterized by bloody droppings, weakness and listlessness. The losses from this disease are largest in flocks that are not provided with ample space. Proper feeding and care will reduce the losses. The provision of ample range and rotation of runs and lots for developing chickens is of value.

Gastric Atony in Ruminants*

B. M. UNDERHILL, University of Pennsylvania

Exactness in the determination of location and character of gastric disturbances in cattle is, as a rule, difficult. While the compartments of the ruminant stomach differ one from the other in structure and function, they are interdependent, and when one is affected usually the other three are involved, the case presenting, we may say, several pathologic conditions co-existing. Aside from disturbances by foreign bodies, cases usually met with are those due to faults in quantity or quality of feed involving commonly the rumen and omasum. The reticulum, due to the peculiar retaining character of its lining, is more often the seat of lesion from penetrating articles as nails, bits of wire, etc. These three prepouches are lined with a thick covering epithelium less specialized than that of the digestive secreting abomasum. Their funcion is associated with that of rumination and the proper mixing and maceration of the food preparatory to its reception by the true glandular stomach. For their proper functioning there must be present a certain vigor of the muscular coat, atonicity of which will, relative to degree, bring about suspended rumination and stasis of rumen contents with decomposition of the food mass and rapid development of gases.

Dietetic disturbances of the prepouches, then, are likely to be of more functional nature than inflammatory, true gastritis having for its seat the abomasum, often of catarrhal form with involvement of the upper portion of the small intestine. Where the affection is intestinal, this may be made evident by diarrhea as a chief symptom. The degree of gas formation in rumen engorgement will be relative to the character of the ingested material, whether rich in water content, as young, standing or freshly-cut clover, or dry roughage, roots and root tops, or the grains. In the event of either there is, in the rumen wall, pressure upon the terminal nerve fibers with consequent stimulation of the musculature to violent contractions in efforts to move onward the offending mass. Unsuccessful in this, the hyper-peristalsis soon gives way to a condition of paresis, the heavier watery portion of the food now settling at the bottom, the dryer material over this, and the gases at the top. Practitioners, especially of the country, are familiar with the clinical picture here presented, as the threatened suffocation, the cyanotic indications of carbon dioxide intoxication and perhaps apoplectoid seizure, due to pressure upon the abdominal arterial trunks with resulting cardiac engorgement and a passive cerebral hyperemia.

Peristaltic insufficiency of the rumen may be primary and pre-disposing or it may be secondary to faults of feeding, prolonged lack of sufficient roughage, foreign body, or perhaps to compression by the uterus in advanced pregnancy. Causes operating to bring it about as an acute condition are numerous, with reference to calves, sudden change of food, as from milk to solid and bulky feed, demanding a degree of gastric muscular activity to which the organ has not as yet become adapted. Over-exertion, strange surroundings, fright, and nervous excitement are other common causes to be mentioned in this connection.

Chronic atony of the rumen, attended with repeated attacks of bloating, less severe than those of acute type, is symptomatic. The cause is to be sought in one or more of a group of conditions which cannot always be ascertained in the living animal. Congenital weakness and flaccidity of the gastric walls or other congenital

*From University of Pennsylvania Bulletin, June 12, 1926.

defect, may be the offending factor, and with this there is to be mentioned any interference with the removal of collecting gases by rumination or belching or their passage by way of the intestines. If such fault is esophageal, attacks may be produced by pressure upon that organ by tuberculous mediastinal glands, and cases have been reported of compression of the lower end of the esophagus by large tapeworm cyst of the liver (echinococcosis). In this connection, we have also to consider the possibility of the presence of sarcomata and actinomycomata, and again, any factor of pressure upon the pneumogastric nerve fibers which supply the stomach compartments. Furthermore, obstruction to the onflow of gas by way of the intestines can readily be brought about by more or less complete occlusion of the passage from one compartment to another by blunt and rounded bodies, such as hair and feed balls which are not uncommonly present in cattle.

Diseases of Swine*

(Report of Committee, U. S. Live Stock Sanitary Association, December 1925. C. H. Stange, Chairman)

IT IS VERY gratifying to note that the most recent statistics from the Department of Agriculture showed that losses of swine from disease were lower in the year ending April 1925, than they have ever been before in the history of the country. The only thing that can conceivably cause a return to the large losses experienced in 1912-13-14, would be the occurrence of a year of hog cholera epizootic. It is felt, however, that this can be prevented if proper sanitary measures and intelligent use of serum are employed. It seems that in connection with hog cholera, one of the most important things requiring to be settled definitely is the question of the advisability of immunizing sucklings.

The question of the so-called breaks following the use of anti-hog cholera serum and virus is still a pressing problem. This being a research question, we believe that the various state and governmental authorities should give careful study to the causes of the so-called "breaks" in immunity; that these "breaks" have occurred there can be no question and the important thing now is to determine positively and definitely the causes for them.

While garbage feeding does not present a major problem in the corn belt, it is nevertheless important in many other sections of the country. Information concerning the dangers of garbage feeding is available and we suggest that garbage feeding plants should have some supervision by animal health authorities. Sanitary officials should be given sufficient authority to prevent outbreaks of hog cholera arising from this source.

The committee heartily commends the careful research work of Van Es and Martin on avian tubercle infection in swine. This question should be pursued until it is definitely solved and a satisfactory method developed for ultimate eradication if that be necessary. Federal and state control agencies and research laboratories should be able to cooperate on this work to the great advantage of the live stock industry.

Mange in hogs is responsible for increased losses each year. We believe that it would be in the interest of economy and strongly urge that control measures be adopted along the lines used in the case of mange in other classes of live stock before the disease

*From Proceedings of the Twenty-ninth Annual Meeting of the U. S. Live Stock Sanitary Association, held at Chicago, December 2-4, 1925.

becomes a greater menace. At the present time some packing plants are reporting as high as forty per cent of the swine coming to their slaughter as being affected.

In connection with the question of sanitation, the following information gathered last year may be of interest. Sixty-five veterinarians selected on the basis of their professional standing and ability were asked a number of questions relating to this problem. Seventy-five per cent were of the opinion that farmers are using better sanitation than previous to the use of anti-hog-cholera serum. All indicated, however, that lack of sanitation is still responsible for much difficulty in swine production, and that improved sanitation would bring about better results in the treatment and control of swine diseases by the profession.

The majority of these men thought that the demonstration method was advisable and in nine-tenths of the cases it was found that personal supervision was necessary in order to get owners to actually apply proper methods.

The efforts now being made to secure general adoption of the McLean County system is commendable. The plan, however, has one handicap in that it entails labor and expense which prevents the average swine raiser from adopting it without a large amount of persuasive effort on the part of the person recommending it.

Practising veterinarians should be encouraged to take a more leading part in campaigns of this kind as in the end the live stock industry will look to the veterinary profession for guidance and assistance in matters pertaining to the conservation of our live stock industry.

PRACTICAL HOG HOUSES

Proper housing is an important factor in the successful raising of hogs. Too often this is neglected, when little expense and effort would be required to provide good, serviceable, well-ventilated houses which give ample protection from cold and admit much needed sunshine.

The same kind of housing for hogs does not apply to all parts of the country, however, says E. Z. Russell, in charge of swine investigations, United States Department of Agriculture. In Farmers' Bulletin 1487-F, just issued by the department, he describes a number of practical hog houses for several sections of the country. One of the chief criticisms which may be made of the average hog house, he says, is that it is poorly lighted and ventilated, if in fact any provision at all has been made for ventilation.

The bulletin contains illustrations and floor plans showing how proper ventilation and lighting is secured in the different styles of houses. A copy of the publication may be secured as long as the supply lasts by writing to the United States Department of Agriculture, Washington, D. C.

Dr. S. J. Schilling of the University of Arkansas, in an article on parasites of livestock, in the June 6th issue of the Ft. Smith Times-Record, points out that a method of preventing the infestation of swine with the thorn-headed worm is to place a ring in the nose of the hog. The larva of the thorn-headed worm are found in the grubs of the June bug or May beetle. Hogs which are able to root will eat many such grubs and become infested with the worms. Hogs with a ring in the nose are prevented to a large extent from rooting and are relatively free from the parasite.

"The men who fail to succeed have two general alibis: 'I'm not a genius,' is one; the other, 'There aren't the opportunities today there used to be.' Neither excuse holds. The first is beside the point; the second altogether wrong."—Charles M. Schwab.

Losses of Pigs Due to Scour

A. T. KINSLEY, Kansas City, Mo.

Scour is one of the most widespread fatal diseases of suckling pigs. It has been estimated that from seven to ten million pigs die annually in the United States from this malady. Some breeders lose from ten to twenty-five per cent ot their pig crop for several successive years from scour. In some instances this disease exacts a toll of from fifty to eighty per cent of the entire number of pigs farrowed. One breeder in Missouri lost over 200 of 300 pigs farrowed during 1925, from scour. The disease has been proportionately more prevalent in the centers of greatest hog population.

Pig scour is most common in young pigs, the majority of cases occurring within the first ten days, although the condition may occur in six weeks old pigs.

Cause

There is a variety of conditions and agencies that are factors in the occurrence of pig scour. Damp, dark, dingy hog houses, insanitary lots and sudden extreme temperature variation predispose to this disease. The exciting cause of the majority of cases of pig scour is improper diet. The feeding of the sow with contaminated or moldy food or fermented slops, probably results in elimination of injurious substances in the milk that will create in the pigs digestive disturbances resulting in diarrhea. Milk from sows affected with mammitis will produce digestive disorders and scour in pigs. Infestation with parasites is another causative factor of this malady. Great care must be exercised in the preparation and administration of artificial feed for orphan pigs, otherwise they will die of scour. After digestive disorders have been established by any of the factors previously enumerated, infection plays an important role. No specific bacterium has been isolated that is responsible for all the terminal infections in pig scour. The B. coli communis, B. suipestifer and other closely related bacteria are the microbian agents most commonly formed in these cases. The B. pyocyaneus and pyogenic micrococci are sometimes demonstrable in the discharges of affected cases and in the lesions of pigs dead of the malady. It is possible that certain microbian agents may attain a pathogenicity and produce a primary infectious scour. Thus the rapid spread of the disease in a litter of pigs and the dissemination from litter to litter and from farm to farm is indicative of the infectious nature of pig scour.

Symptoms

Pig scour is characterized by diarrhea. The evacuations in the beginning are pasty in consistency and of a gray or slate color. Later the discharges become more watery and have a very disagreeable odor. The pig's tail and hind parts become soiled. The longer the disease persists the more fluid and fetid are the discharges. The appetite is good in the beginning but the affected pig soon loses all desire for food. There is abdominal pain evidenced by the affected pig kicking the belly, squealing, grunting and by restlessness. The animals have a dejected appearance, standing with head down, tail drooping, tucked abdomen and rough coat. Emaciation is rapid and the affected pig becomes weak and dies of exhaustion.

Lesions

There are no specific lesions that characterize pig scour. There will be soiling of the tail and hind legs, tucked abdomen, rough coat and unless the disease is very acute there will be marked emaciation. The digestive tube may be practically empty. Any content in the

intestine will be fluid and probably putrid. There may be areas of congestion in the intestinal mucosa. In subacute or chronic cases there may be inflammation and ulceration of the mucosa in the stomach or intestine.

Treatment

The success of treatment of pigs affected with scour will depend upon the vitality of the pig, the cause of the malady, and the stage of the disease. As a rule pigs affected with scour have little vitality and therefore do not respond to treatment. The usual cause of this disease is improper diet and those pigs that are fairly strong and are in the early stages of the disease will survive if the diet is corrected. Medicinal treatment is usually of little value. The use of castor oil after correcting the diet serves the double purpose of eliminating the bowel content and soothes the irritated mucosa but requires time and care to administer. If the diarrhea persists after providing the proper diet for the sow and the administration of castor oil, small doses of bismuth subnitrate may be used to advantage.

When scour is due to infection with B. suipestifer or related microbes, the use of a bacterin made from cultures of those organisms will be of value not only as a preventive agent in exposed pigs but also a curative agent in those affected.

Generally considered scour in pigs is primarily of dietary origin and its prevention depends upon providing the proper diet.

Unsolved Cases

FRANK M. WHEATLEY, Fort Qu'Appelle, Saskatchewan

It is not only a pleasure to contemplate past cases, but it is also instructive when considered in the light of present day treatments.

Some few years ago, in the late fall, one of my clients telephoned that he had a very sick mare, a great favorite that he did not wish to run the risk of losing, so would I come at once.

I arrived at his place and found the mare with a temperature of 106° F., a full bounding pulse, highly injected visible mucous membranes, quickened respiration, head and ears drooping and general dejection. The only history was that she had lagged a little during an afternoon climb and refused her evening meal.

Febrifuges, diuretics and stimulants were administered. For two days the mare seemed to improve, but on the third day pulmonary complications appeared. Unfortunately at this stage the owner on the advice of a quacking neighbor administered a stiff dose of linseed oil with the object of "clearing the mare out." This he did very effectually for superpurgation ensued and the mare rapidly wasted in spite of all we could do. She died on the tenth day.

Some days after the mare died, two more horses in the same barn became sick. Practically similar symptoms appeared in their cases with the exception of purgation—rapid onset, a day or so of ailing, then decline. The appetite did not seem affected and they ate until the last. In their cases, however, pulmonary trouble seemed more accentuated. They died on the tenth day. Postmortem examination revealed hepatized lungs, distended pericardial sacs and some inflammation of the alimentary tract.

I diagnosed intermittent fever of a virulent type but could attribute no cause for it, not having seen anything quite similar during the war, even when associated with hundreds of cases of shipping fever and allied complaints.

During this time two neighboring farmers each had a horse that developed symptoms exactly resembling the ones described, but curiously enough both these subjects responded to my treatment and recovered.

A week after the third animal died, three more came down with the disease. I advised the owner to call in a neighboring veterinarian. This he did and in consultation a slightly varied treatment was devised. Results were similar, and before these animals died still another veterinarian was called in consultation.

The owner having lost six horses communicated with the Provincial Health of Animals Branch. They referred him to Saskatoon University, a member of the veterinary staff of which was a specialist on such matters and was even then investigating possible causes of similar equine diseases. To be brief, the remaining six horses of this client shortly afterward sickened. The university veterinarian was called, came, investigated, consulted and treated, but all to no avail and the six animals died.

The farm pastures were combed for poisonous weeds; food and water supplies were carefully checked over; but no solution of the cause was arrived at. The owner was advised to destroy his barn, which was a collection of old log buildings, also to use fresh water supplies and change his pastures. These things he did, and to date has had no recurrence of the trouble.

I am now inclined to classify the disease as of a paratyphoid type and should be interested to learn the opinions of other readers more experienced and better informed who might possibly throw some light on the subject.

Another Curious Case

The subject was an Airedale bitch, one year old. Late last fall the owner reported her as having a series of convulsions. These consisted of running in a circle, barking furiously, banging into any kind of object and foaming at the mouth. These attacks continued for about five minutes, when the animal would subside into a perfectly normal condition.

Four of these wild periods were allowed to occur before the owner consulted me, and then, as I was not able personally to examine the bitch, I suggested the trouble was probably of parasitic origin and prescribed a reliable vermifuge to be given. From that time until recently (some ten months) no further symptoms occurred, and it was surmised that the cause had been removed and so the effect had ceased.

However, a few weeks ago the bitch underwent another convulsion, the owner telling me that her great ambition seemed to be to acquire altitude, as being in the house she leapt on to chairs and tables and endeavored to scale the wall. This attack I also did not observe, but had examined her carefully a few days previously and found her apparently in normal health and condition. The owner in this instance had himself administered a dose of cascara sagrada, and for a few days all was well.

One day I happened to be out in this part of the country and was talking to the client in the field where he was drilling. The bitch had followed him all morning and was nosing around as usual, when suddenly she commenced to bark and run in a circle around the outfit of horses and machinery. The owner remarked, "She's got 'em again." As she careered around apparently quite demented, I observed her slavering profusely and running blindly, but not so blindly as to prevent her endeavoring to climb on the drill by repeatedly banging against the wheels and rising on her hind legs.

After several attempts she worked round to the steps and leaping on to the drill box, to our amazement, she leapt from there on to the backs of the horses. This action, of course, started the team and they broke away. The bitch fell or rolled under the drill before the team was stopped. Although wounded and

winded, she got up and began her circling again. At this juncture the owner grabbed a large monkey wrench and wrote "finis" to the animal's career.

On close examination I noticed she was approaching estrum, but otherwise an autopsy revealed no reason for her strange behavior. Again in this instance, can any reader throw light on the subject? Was it connected with the estrus cycle or of cerebral origin?

Cecal Abligation in Fowls

A. J. DURANT, A. M., D. V. M., Poultry Pathologist, College of Agriculture and Experiment Station, University of Missouri

Cecectomy in fowls is a difficult and unsatisfactory operation, as I have found in the several attempts I have made to perform that operation on chickens and turkeys for the removal of the cecal habitat of coccidia and of the parasitic worm Heterakis papillosa which some investigators regard as having an important if not an essential relation to the transmission of "blackhead" in turkeys. The difficulties encountered led to the development of another operation designed to serve the same purpose. This operation is a comparatively simple one, merely that of tying off the cecal pouches at their junction with the main gut. The ceca are left in place without any surgical disturbance to the mesenteric attachments and blood supply. This operation prevents the further passage of food materials, feces or parasites into these intestinal diverticula. The ligation ultimately effects a complete severance of the ceca from the main gut, and the stumps produced by the ligature wounds heal over in most cases so perfectly as to prevent any leakage from the functional gut or from the detached ceca. Any fecal matter that may be retained in the isolated ceca does not seem to cause any serious disturbance to the health of the fowl. And, as judged by the egg laying capacity of the birds subsequent to the operation, the ceca do not appear to be essential organs of digestion or of absorption. It is not, however, the physiological aspects of this matter, nor the possible applications of the operation to the prevention of special parasitism of fowls, ("blackhead") that concern us at the present time. The purpose of this paper is to describe the surgical technic of the operation, and to show that no harmful surgical sequelae attend the operation when it is performed in a proper manner.

The word "abligation" is used to differentiate this operation from cecectomy or cecotomy; since the ceca are not "cut out," nor "cut into," but are "tied off" and left in position. The word is not found in the medical dictionaries, for the good reason, perhaps, that in both human and veterinary surgery such an operation on the alimentary viscera has never been attempted or at least so rarely that a special name has not been applied. The word, however, is not a newly coined word; but a very old and almost obsolete English word, of Latin origin, which is here revived for appropriate use.

Mention was made that the operation of cecectomy in fowls is difficult and unsatisfactory. The reasons for this are that in fowls, relative to the size of the animal, the ceca are much larger and the blood supply much greater than in the dog, upon which animal cecectomy is a comparatively simple operation. Moreover, in the fowl the ceca are paired and are curved upon themselves. These anatomical peculiarities make it difficult to cut away the cecal mesentery and to restrain the resulting hemorrhage. Be-

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Fig. 1. Normal ceca of fowl

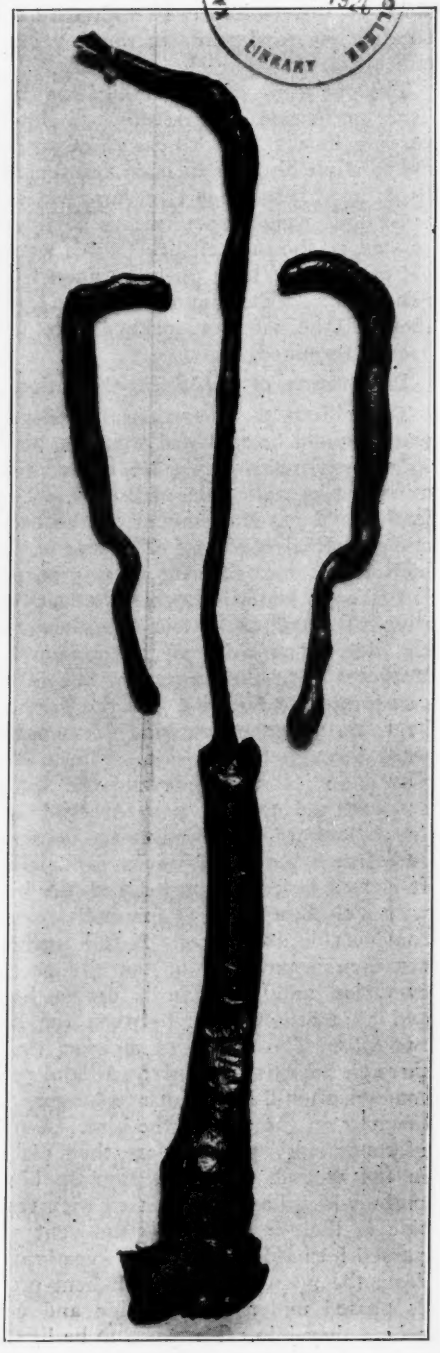

Fig. 2. Abligated ceca of fowl

sides, two operations are required to dececalize the fowl while in the dog only one operation would be necessary.

The operation of cecal abligation has been performed successfully on fowls varying in age from chicks three weeks old to adult birds, both male and female. Birds most suitable in size, however, are those that weigh from twenty to thirty ounces. In the case of turkeys five weeks old poults have been operated upon with good results. The limits of size and age that will be safe for turkeys have not been determined.

Preparation of Birds for Operation

To facilitate the operation all food and water should be withheld from the birds for twenty-four hours. No other preliminary preparation is necessary. This fasting reduces the amount of the cecal and intestinal contents. The site of the incision for exposing the ceca is on the left side of the bird between the last two ribs—the same as that in caponizing on left side. The point of junction of the two ceca with the large intestine is located opposite this site, and the parts to be operated upon are easily drawn upward through the incision. The bird is placed on its right side and the wings are fastened with a loop of cord and drawn forward, and the feet are fastened and drawn backward, as in caponizing. It is well to cover the head of the bird with a cloth or hood, as this will prevent considerable struggling. A few feathers are then removed from the site of the operation and the skin is drawn back and an incision made between the last two ribs. The length of incision, made through the skin, muscular wall and peritoneum, should be an inch or longer, depending on the size of the bird. A pair of caponizing spreaders are then placed in the wound. A small smooth, blunt probe which has been greased with vaseline is then inserted into the vent and passed forward to where the ceca branch from the main gut. A small bent probe is passed under the intestine and ceca where they join. The parts to be ligated are then drawn gently up through the incision in a convenient position for operating. If this is done carefully, but little hemorrhage will occur as only a few small blood vessels will be ruptured.

A small thread carrier threaded with a 20-day chromic catgut ligature is then passed between the main intestine and one of the ceca where it branches. The size of the suture will depend somewhat on the size of the bird to be operated upon; for adult birds size No. 3 is most suitable. For smaller birds a smaller size ligature should be used. The thread carrier is then unthreaded, leaving the ligature around the cecum. The cecum is then tied off at a point close to its junction with the main gut. Care should be taken to draw the thread tight enough to effectually close the passage into the main gut, but at the same time not tight enough to cut through the outer cecal coat. The same procedure is carried out upon the other cecum, and the intestines are then gently replaced. It is unnecessary to stitch up the incision in the abdominal wall since the skin-flap will cover the opening when the bird is released.

No special after care, as a rule, is necessary except perhaps to modify the diet for a few days and to reduce the wind puffs which may occur. These wind-puffs do not occur with any greater frequency than in caponizing. Aside from the wind-puffs, the birds usually make an uneventful recovery. Adult birds as a rule appear perfectly healthy within seven days after the operation; and those that are in laying condition, at the time of the operation, will be laying again in ten days or two weeks. In small chicks the recovery from the operation is much more rapid unless some unusual complication occurs.

Care should be taken to avoid the mistake of ligating the main gut for one of the ceca; since anterior to their junction the three organs are at times very similar in size and color. In the experience of the writer this happened in two cases

and death of the birds resulted in four or five days from obstruction of the intestines. This error may be avoided by keeping in mind the fact that each cecum has a rather distinct neck where it branches from the main gut and that the two are located nearer each other on one side of the main gut than on the other.

It will be well to emphasize the fact that if the ligatures are not drawn tightly enough to effectually obliterate the passage, the cecum will function again after absorption of the ligature. On the other hand, if the ligature is drawn too tightly there is danger of cutting through the gut and causing leakage and peritonitis. The surgical knack of tying the gut properly can, however, be soon acquired by each operator from actual experience.

The instruments used in the operation should be sterilized and proper asepsis observed otherwise peritoneal adhesions may result that will hinder the normal functioning of the intestines. Leakage from the ligature wound, however, is probably the most frequent cause of peritonitis when this complication follows the operation.

Changes Produced in the Ceca by the Operation

Since the blood supply of the ligated organs is not cut off they do not become necrotic but gradually shrink from disuse. The ligatures gradually sever the ceca from the main intestine, and the separated stumps will be found healed over, leaving the ceca as free hanging sealed tubes, suspended by their mesenteric ligaments and slight cicatricial bands adhering to the surgical stumps.

Fig. 1 shows the normal ceca and contigious parts of the small and large intestine of an adult hen. The mesentery has been dissected away to show more clearly the cecal sacs and their constricted necks at junction with the main gut.

Fig. 2 shows the corresponding organs of an adult male bird in which the ceca were abligated. In this case the mesentery has also been dissected away to show more clearly the changes in the organs at the site of operation. The ceca, as will be observed, are completely separated from the main intestine, and the cut surfaces are healed over, leaving the ceca as free sealed tubes.

A slight leakage will sometimes occur from the cecal side of the ligature; this, however, does not usually produce any serious complications and is readily encapsulated. If leakage occurs from the proximal side of the ligature wound, that is, from the main gut, the dangers are greater; such leakage, however, is comparatively rare.

As to whether this operation will prove to be profitable to the veterinary practitioner, even if it should become a routine practice in turkey raising, will depend upon the rapidity with which the operation can be done, and the fee per bird. With the present technic and limited experience, the shortest time in which I have done the operation on a bird is one and a half minutes. It is probable, however, that with improved technic, and greater skill from experience, many practitioners will be able to do this operation more rapidly. It is doubtful, however, whether it can ever be done as rapidly and as safely as caponizing.

FOX DISEASE RESEARCH

In the June issue of the American Fox and Fur Farmer, Dr. R. G. Green reports on the recent work of the distemper and research committee organized by fox breeders. He states that there have been discovered thus far two definite infective agents capable of killing foxes upon injection. One, a group of bacteria belonging to the Salmonella, which upon injection into animals produces a fatal infection with intestinal lesions and enlargement of the spleen; the second infective agent, a filtrable virus, which produces a generalized infection, the virus being found in the blood. The relative importance of these two infections in producing epizootics on ranches is not yet known.

Diaphragmatic Hernia—A Cause of Ascites in a Dog

F. F. TURLA and L. M. YUTUC, Manila, P. I.

So far as the writers have been able to review the literature on diseases of dogs and other domesticated animals, not a single case of diaphragmatic hernia causing ascites in animals has been mentioned. In presenting this short discussion, therefore, our object is to put on record one such case in a dog which occurred recently in our veterinary clinic.

Clinical Record

On March 18, 1926, a smooth-haired Fox Terrier male dog, about two years old, was brought to the hospital for consultation. The owner complained that once in a while during the time the animal was taking his meal, she observed him uttering a peculiar cry as if in pain. One of his hind legs was also noted in an abducted position, at the same time curving his body toward the left side. She claimed that the animal was run over by an auto about a week before but apparently no injury happened at the time of the accident, as the dog appeared normal in every way. However, she was not absolutely sure.

While the history was being taken, the animal was placed on the table and examination made. The temperature was 38.6° C. Pulse and respiration were accelerated. Percussion and auscultation revealed nothing abnormal. Palpation of the thorax as well as the abdomen revealed neither pain nor enlargement. Mucous membrane was slightly injected.

With the history given plus the symptoms observed, we could not arrive at a definite conclusion. In view of this, we asked the owner to leave the dog, assuring her that the next day the animal would be re-examined. We made her understand that the acceleration of pulse and respiration might be brought about by the trip which the animal made. At last, she consented to the proposition.

The following day the dog was examined more closely. Temperature 39.7° C. Respiration was shallow and somewhat labored. The head and neck were held extended in a horizontal position. Percussion and auscultation showed nothing peculiar, save an exaggerated vesicular murmur. No friction sound detected. Palpation over the same region revealed no pain. The abdominal region was likewise manipulated but no abnormalities were determined.

The diagnosis was reserved. But, with nothing to say when the owner called up, we put up a probable diagnosis. We called the case some kind of a pleurisy.

The treatment for this ailment was instituted. White liniment as a counter-irritant was rubbed with friction over the thorax and a flannel bandage applied loosely. Fluid extract digitalis was given as a cardiac stimulant. As diuretics potassium acetate and urotropin were administered. Every second day magnesium sulphate was given as a laxative.

On March 30, 1926, we observed the animal with no improvement at all; instead the respiratory act became more aggravated and the animal stayed most of the time either on the abdomen or sitting on his haunches. His appetite was capricious; abdomen seemed to have increased in size. We decided to hold another examination.

The following were observed: Enlargement of the abdomen especially prominent when the animal was in a standing posture. The flanks were sunken and the transverse processes of the lumbar vertebra outstanding. Mucous membrane was cyanotic although not marked. Temperature was normal. On raising the hind quarters the respiration was made more dyspneic and the animal appeared as if gasping for breath. By placing the left hand on the side

of the abdomen and then gently tapping the other side by the right hand a plain fluctuation of the fluid was felt. Changing the position of the animal the horizontal line of dullness was shifted. Viewing the animal anteriorly as well as posteriorly, we noted a symmetrical expansion of the abdomen which appeared like a suspended bag of water.

From the symptoms noted it was a case of ascites.

Our treatment given in the foregoing discussion was not varied, save an addition of potassium iodid as a resorbent. We kept on this line of treatment until the middle of April, when we were induced to resort to other means. Paracentesis abdominis was decided on at last for the case, as the accumulation of fluid tended to interfere with action and functions of the abdominal and thoracic organs.

We notified the owner about the condition of the animal, saying that we had to resort to radical measures of alleviating the disease as palliative treatment had been entirely fruitless. So far as prolonging the life of the animal, we gave a fairly favorable prognosis, and in effecting a permanent cure there was hope inasmuch as the subject was young. However, we did not assure her that the condition would not recur. Also, that the danger of collapse from shock might come out any time during the operation, was not overlooked. In spite of the unfavorable prognosis given, she submitted the animal to the last ordeal.

The next day the animal was operated on. After observing the preliminary technic for this particular operation, the dog was punctured. The fluid obtained was about 500 cc. It was reddish in tint and fairly clear. Soon after the operation was done, the animal appeared greatly relieved. The appetite was partially restored.

On April 26, 1926, the animal appeared again in great distress, taking no notice of the surroundings. The abdomen seemed grown in size more than formerly. He was tapped again and about 800 cc. of fluid withdrawn. We kept on doing the same operation when found advisable. Still the condition recurred. The formation of fluid seemed faster than we could remove it, and so we gave up the case and declared it as incurable.

We suspected that there must be a chronic disease going on, as, for example, compressed mesenteric vessel, chronic peritonitis or cirrhosis of the liver, which we could not possibly diagnose clinically.

On May 16, 1926, the animal was destroyed by injecting a fatal dose of strychnin sulphate subcutaneously.

Post Mortem Findings

External examination: Rigor mortis present as indicated by the stiff condition of the cadaver with all the extremities extended. The mouth was open with the corners drawn back. The eyes were open. Prepuce and hair around were smeared with light yellow urine. The anus and perineum were soiled with feces. The coat was somewhat lusterless and short.

Internal examination: Upon opening the abdominal cavity a large quantity of fluid rushed out. It had the same color as that obtained in the operation, although with numerous flakes of coagulable lymph.

The serosa of the intestines and the stomach was smooth and dull white. The mesentery was examined for obstructions in the course of blood vessels, but none found.

When we proceeded to locate and examine the liver our attention was at once attracted by a big rent in the diaphragm. It was as large as a peso and almost triangular in outline. Inspection revealed that some of the lobes of the liver went through it into the thoracic cavity. The edges of the rent were smooth and rounded, showing that the case was long standing. The herniated lobes were the left central, the papillary and about three-fourths of the left lateral including the gall bladder. The hernial ring was located in the right portion of the diaphragm about a centimeter from the xiphoid and one-half centimeter from the costal attachments of the diaphragm. The lobes were fused with each other and appeared like a large single lobe. They were bluish brown in color with gray and thickened capsule. They were very much enlarged and the

edges blunt. The lobes were cut into thin parallel sections. Incision margin bulged out. Cut surface revealed bluish red blood oozing out with the central veins engorged. The capsule was stripped off with difficulty and the parenchyma was very firm, indicating an increase of interlobular connective tissue. The other lobes were found in situ. They were reddish brown with edges sharp. The parenchyma was firm and incision margin even. The hepatic veins supplying the dislocated lobes were found twisted and this resulted in a chronic venous congestion that eventually led to the effusion and collection of fluid in the abdominal cavity. The gall bladder contained greenish yellow fluid.

The spleen was divided into two independent parts. The ruptured ends were healed up nicely. It was light red and soft in consistency except near the ruptured ends where it was firm due to the cicatrization of tissue. Cut surface was flat and incision margin did not bulge. The pulp was brownish red and the capsule adherent and tough. Dark red spots were seen in the gastrosplenic ligament from a pinhead to as large as a millet seed.

The kidneys were bean-shaped, firm, and grayish brown. The incision margin was even. The capsule was thin, smooth, opaque and loosely adherent to the parenchyma. Cut surface showed faintly myriads of minute round bodies which were the glomeruli.

The peritoneum appeared smooth, moist, and glistening except in two places where we noted groups of dark red tiny spots.

The thoracic cavity was also laid bare. It was partly filled with fluid. This must have originated from the same diseased organ. Some of the lobes of the lungs were in a state of collapse. They were dark red in color and of fleshy consistence. The apical lobe of the right lung was connected by a band of connective tissue to the thoracic wall. This showed that pleurisy suspected as mentioned above possibly took place. They looked almost like fetal lungs. Pieces placed in water did not float. The pleura of the thoracic wall was moist, glistening and dull white.

The heart was opened. The valves were not thickened. The auricles and ventricles were filled with coagulated blood.

Pathologico-Anatomical Diagnosis

1—Ascites.
2—Diaphragmatic hernia.
3—Cirrhosis of the liver.
4—Rupture of the spleen.
5—Hydrothorax.
6—Local chronic peritonitis.
7—Compression atelectasis.
8—Asphyxia.

Epicrisis

The traumatic injury which the animal received from the auto accident as the owner mentioned in the anamnesis was probably the cause of the diaphragmatic hernia, the rupture of the spleen and possibly the local chronic peritonitis. The other conditions occurred as secondary diseases, except asphyxia, from which the animal died.

Pruncha and Brannon report that typhoid bacilli remained viable in ice cream kept at a temperature of 8° F. below to 8° F. above zero for two years and four months.

Stringer concludes that Mercurochrome 220 used intravenously is not a panacea in septicemia and that its use is not without danger. Its low toxicity recommends its use in extreme cases with a certain margin of "fight" still present.

Oxygen is detrimental to some of the single-celled plants and animals, according to Dr. Cleveland of Harvard University Medical School. The doctor has demonstrated the destructive property of oxygen in termites infested with protozoa and on foodstuffs contaminated with bacteria, yeast and molds. It may be possible to use oxygen sterilization, not only in the preservation of foods, but also in the treatment of infective diseases as well as those caused by protozoa.

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Purpura in the Dog

OSCAR SCHRECK, New Haven, Conn.

Definition

Extravasations of blood, in the form of points, petechiæ, or ecchymoses, are not uncommonly observed beneath the surface of the skin in various forms of disease, and under many other conditions, and are often termed purpuric. Not infrequently these subcutaneous extravasations (especially if due to constitutional disorders) are associated with similar extravasations into solid organs, beneath the serous and mucous membranes, and with more or less abundant escape of blood from the surfaces of these membranes. These hemorrhages may be of various sizes. When small, they are called petechiæ, when larger, they are called ecchymoses. There is no proof that purpura is a disease of the blood in the small animals. Such extravasations are especially found in the case of distemper, obstructive heart and liver disease, as secondary disease, also following pyemia, and in embolism. They further occasionally complicate certain skin diseases, more especially some of the forms of erythema and urticaria (very rare, however) and may be produced in certain regions by mere exposure to atmospheric influences. But to none of these affections, however severe they may be, can the term purpura be properly applied, in the small animals. Purpura in the strict sense of the term (the morbus marculosus, Werlhofii, of the Germans) is the name given to an affection characterized by such hemorrhages as have been above specified, but unconnected, so far as we know, in the small animals, with any local mischief or any general specific disease.

Causation

Purpura is not limited by climate, sex, or breed. It appears to be strictly sporadic. The causes of purpura and the conditions under which it arises in the small animals, are exceedingly obscure.

In some instances, the disease appears to arise from causes of a debilitating nature, calculated to impair the efficiency of the processes of sanguification, while, at the same time, there is a want of material out of which the blood is made. Hence, unwholesome, indigestible, and scanty food, confined and impure air, continued fatigue, and the debility induced by various diseases, as distemper, have been ranked among the causes of purpura. It occurs at all ages, but mostly in young animals of both sexes. It may be observed among animals that are sickly, underfed, or surrounded by unwholesome sanitary conditions, but it is also met with among the robust and healthy looking, and those whose hygienic and other circumstances appear to be unexceptionable. An infectious origin is not unlikely. Purpuric spots are not infrequently seen with valvular diseases of the heart, cirrhosis of the liver, and various forms of fever. Distinct exciting causes, if such exist, are obscure. Fright, severe coughing fits, and epileptic attacks are said to have induced it. There is no doubt in my mind but that the enfeebled condition of the vessels often depends upon a state of general debility, either hereditary or acquired.

Symptoms

The onset may be marked by moderate fever (102° to 103° F.). In some cases purpura presents an acute form, but is generally unattended with fever or severe constitutional disturbances, unless from loss of blood the strength is materially impaired. This loss of blood happens in some cases into the loose connective tissues of the body or from the lungs, and still more frequently from the bowels or urinary organs. Under these circumstances the pulse, which otherwise is apt to preserve its normal frequency, becomes very rapid, but until exhaustion commences to tell on the nervous system, not as a rule long before dissolution—the ani-

mal remains alert, and cerebral or spinal symptoms are absent.

There may be or may not be hemorrhage from the mouth, but almost always bluish red hemorrhagic spots about the size of a ten cent piece to a quarter dollar are visible under the breast, in the fore and hind leg pit, on the abdomen and inner surface of the thighs, and are wholly uninfluenced by pressure.

Desquamation never follows, and once formed a spot does not increase in size, except by fresh hemorrhage in its vicinity. They do, however, gradually change their color and fade. They are also by no means confined to the above mentioned situations, and not infrequently extravasations take place into the eyelids, beneath the conjunctivæ, and beneath the mucous surface of the tongue, lips, gums, and other parts within the mouth of the animal. The gums are not soft and spongy, as in scurvy, nor do we observe the same weakness of the animal body. Moreover, although like scurvy, the disorder may be benefited by iron, bitter tonics, and the mineral acids, it is not controlled like scurvy, by lemon juice—in fact, by agents that are most decided antiscorbutics. The fact that there is no itching, no desquamation, no suppuration or discharge, and no change in purpuric spots upon pressure suffices to distinguish them from the eruption of any form of skin disease.

Nasal hemorrhages and loosened teeth, particularly the incisors, may be frequent. In many instances, the preliminary symptoms are wholly wanting and the appearance of the petechiæ marks the commencement of the disease. One case coming under my observation, was wholly without external hemorrhage; in others, this is the most prominent symptom, such as blood escaping from the nostrils, gums, and other parts of the mouth, conjunctiva, stomach, bowels, kidneys, and urinary passages, rectum and vagina, sometimes excessively, so as to occasion immediate alarm, but as a rule in small quantities.

Anemia and its associated phenomena develop in severe cases.

Anatomical Characteristics

The spots upon the skin are found to be due to blood extravasated immediately beneath the cuticle, or in the texture of the cutis itself. Blood is also seen occasionally in the subcutaneous cellular tissue, forming extensive ecchymoses. This may be washed away, leaving the vessels without any marks of injection, or other evidence of inflammation. Petechiæ similar to those on the skin are found in the mucous membrane of the alimentary canal, and sometimes though more rarely, and less numerously, upon the serous membranes, as the pleura, pericardium, and peritoneum. Extravasation of blood sometimes exists also in the lungs, in the parenchyma of the viscera, and in the substance of the muscles. In several post-mortem examinations the blood was found to be remarkably fluid, even in the tissues into which it was effused in considerable quantity.

Course, Duration, and Termination

The whole course of the disease includes the prodromal period, the purpura simplex, the period of hemorrhage, and the subsequent anemia. The duration is influenced materially by the number and amount of hemorrhages. An ordinary case will last two or three weeks, but when there are repeated hemorrhages, the disease may continue for several months. Although most cases recover, death sometimes happens from exhaustion, from internal hemorrhage, from some intercurrent malady, and from perforation of the bowel.

Diagnosis

Purpura may be confounded with scorbutus, hemophilia, progressive anemia, cerebro-spinal meningitis. From scurvy it is differentiated by the absence of changes of the gums, of the indurations of the subcutaneous areolar tissue and of the muscles, of the hemorrhagic inflammation of serous membranes, etc. From hemophilia, the distinction is made by reference to the history, especially heredity, by the period of the animal's life, by the bleeding from trivial wounds, so characteristic of hemophilia, and not of purpura. The distinction of purpura from progressive anemia rests on the fact that in

the former the anemia is produced by the bleeding, in the latter the bleeding comes on afterward and is due to the poverty of blood. The initial symptoms of cerebrospinal meningitis may be almost identical with those of purpura; purplish spots, pain in the joints, by palpation, with some slight feverishness, but in a day or two the occurrence of nervous phenomena decides the question.

Prognosis

In uncomplicated purpura the prognosis is good, but when venous thrombosis, diarrhea, or an incurable organic disease exists, the animal's life is endangered by the liability to hemorrhage from mucous surfaces and the occurrence of extravasations into the serous cavities of the brain. Anemia and dropsy are often causes of small animal deaths. Relapses are not uncommon.

Treatment

The severer cases are unfortunately apt to go from bad to worse whatever treatment be adopted. The usual treatment consists in the administration of the mineral acids, especially the sulphuric, and the preparation of iron, especially the tincture of chlorid. With these remedies must be conjoined a suitable dietary, fresh air, sunshine, and moderate exercise. If constipation is noticed, the most appropriate laxative in small animals suffering from this disease, is sulphate of magnesia with dilute sulphuric acid. If hemorrhages that are threatening come on with a strong pulse, excitement, and uneasiness of the animal, digitalis, quinia, and ergotin are the appropriate medicaments. If there be weakness and debility, quinin and alcoholic stimulants moderately should be prescribed for the animal. The local means for arresting bleeding consist in sub-sulphate of iron, tannin, alcohol, ice, or again it may be hot water, which is sometimes more effective in the small animals than cold. For the anemia iron should be pushed. Turpentine and gallic acid are also indicated. Cold drinking water should be given, and renewed quite often. If asthenia be extreme, it is absolutely necessary to push alcoholic stimulants. On the whole, tonic treatment is indicated also in those animals who have a tendency to purpura and in those who are convalescent from it. When hemorrhage from the lungs occurs, the treatment is the same as in other forms of bronchial hemorrhage. Recently, small doses of mercury have been given, and apparently effected a cure in some cases.

℞

Olei terebinthinæ	ʒ i	4.00
Sacchari	ʒ iv	16.00
Acaciæ	ʒ iv	16.00
Aqua q. s.	℥ iii	90.00

Misce fiat emulsion.

Sig. Teaspoonful after feeding.

Indications: Useful in animals with debility and vascular relaxation.

℞

Suprarenali sicci	ʒ i	4.00

Pone in capsules No. XXIV.

Sig. One to three capsules t. i. d. after feeding.

Indications: Used in purpura with vasomotor relaxation.

℞

Potassii iodidi	℥ i	32.00
Aqua bullientis q. s.	℥ i	30.00

Sig; Five to twenty drops in milk, t. i. d. after feeding.

Indications: Useful in purpura.

℞

Calcii chloridi	℥ iv	15.00
Aqua	℥ vi	180.00

Misce.

Sig. Teaspoonful to a tablespoonful in water t. i. d.

Indications: Used in severe and obstinate cases.

The animal should be fed freely with gelatin.

Case Reports

A Great Dane pup, eight months of age, had always been in perfectly good health, was still running around with the bitch, who was a healthy strong animal, and had two other healthy pups at this time. The stud dog was also remarkably strong and healthy. This pup, without any noticeable previous symptoms, suddenly developed, what I diagnosed as purpura. Large ecchymoses appeared upon the inner aspect of the hind and front legs, about the size of a pea; the ani-

mal rapidly failed in strength, and died in forty-eight hours. There was no hemorrhage from the mucous membranes.

In a Boston bull dog, the skin of the inside of the extremities showed numerous ecchymoses of various sizes and of a dark red and purple color. On the abdomen on both sides of the nipples were two large ecchymoses, also smaller ones scattered about between the larger ones. On postmortem examination, nothing was found except a slight atelectasis of the lower lobes of both the lungs, with adhesions of the pleura, and a slight degeneration of the heart, liver, and left kidney, and enlarged mesenteric lymph glands.

Poisonous Plants

L. H. PAMMEL, Dept. of Botany, Iowa State College

RAGWEED AND HAY FEVER

From time to time I have had a few notes on the subject of hay fever. One of the very common plants producing hay fever is the small ragweed which is extremely abundant in many of the northern states, said to be injurious to cattle, in what way, it has not been definitely determined. Also, this same plant produces pollen which causes hay fever. The value of our pastures is materially reduced by the presence of ragweed.

Dr. W. A. Evans recently had the following item in the St. Louis Globe-Democrat concerning hay fever:

"Hay fever is due to poisoning by the pollen of certain plants. There were those who thought that nervousness, imagination, and all that group of mental states were responsible. The fact is that nervous instability is an element in hay fever, but it is not a chief cause. It is merely a contributory cause, and not one of great importance.

"There were those who held it was caused by abnormalities in the nose. The fact is that these abnormalities are of some consequence; but they are not a chief cause. They merely contribute. There were those who held that hay fever was nothing more than asthma. It is a variety of asthma. After a person has had hay fever a long time, he may become asthmatic. It is a fact that the form of asthma known as hay fever is due to pollens, but some bronchitis due to bacterial infection complicates it, and in time a true bronchial asthma may develop out of what, in the earlier years, was a true hay fever.

"The scientists are still arguing whether hay fever is anaphylaxis or an allergy, but that is a question which concerns them and which you and I can forget about while we go on with our job.

"And what is our job? The job of the fellow who is subject to hay fever? To find what causes it, to find a preventive and to find a cure and use that if necessary and to help in the removal of the cause.

"Hay fever is caused by pollens. But which pollen causes the disease in any particular case, since there are so many pollens and some harm some people and others other people? Skin tests show the pollens which harm given individuals. These tests are easily made and the material for them can be obtained by any physician. Even if skin tests cannot be made the only important causes of hay fever in any given locality are about six in number. These, roughly speaking, are divided so that about two are in each of three groups.

"The old rose asthmas are due to tree pollens. Next as the season goes on, are the hay fevers due to grass pollens; and finally, the great group of hay fever starting generally in August and due to weed pollens. Many manufacturers now give away a list of the important pollens and

when they are abundant in the various sections of the country. I have before me as I write, one such list, giving the information for each state and for each type of soil. Generally speaking, oak and elms are responsible for rose fever, timothy and June grass for the hay fever of May and June, and common ragweed and giant ragweed for August and September hay fever."

SWEET CLOVER

Mr. Thomas P. Kerl, of Coeur d'Alene, Montana, asks for information about sweet clover poisoning. I referred the letter to Dr. Charles Murray, who writes an interesting reply. I take the liberty of using it:

"Replying to your letter of April 12 enclosing letter from Mr. Kerl, will say that in Vol. 17, No. 5, of the Journal of the American Veterinary Medical Association, is an article by Dr. Frank W. Scofield of the Ontario Veterinary College describing the disease more or less prevalent in Canada which from his investigations seemed to be caused by cattle feeding on moldy sweet clover. He gives the results of feeding experiments, also the pathology of the condition produced. As he describes it this disease somewhat resembles hemorrhagic septicemia and also blackleg. Hemorrhages in the subcutaneous tissue and internal organs are commonly observed. The outstanding feature of the disease is its ability to so affect the animal that coagulation of the blood will not occur and animals injured may readily bleed to death or may have internal hemorrhages from ruptured blood vessels.

"Doctor Roderick of the North Dakota Station, gave a report at the meeting of the Livestock Sanitary Association in Chicago this winter on his study of the disease. His findings were quite the same as Dr. Scofield's. I think some work has also been done at the Ohio State University.

"We have observed only one case of this kind. This was in a herd of dairy cattle belonging to Mr. Conn of Boone, Iowa. Dr. Covault and I visited that herd about a year ago. There were already some six or seven of the animals dead, and before the trouble was stopped I think the loss amounted to some three or four adult cows and ten or more heifers. I had the opportunity to autopsy one of the dead heifers and found the condition quite as described by both Dr. Scofield and Roderick.

"A very interesting although unfortunate thing occurred while we were visiting this herd. One of the cows had a growth of membrane across the vagina, and Dr. Covault was asked to operate and to sever this membrane. The cow was in apparently normal condition and without thought of her possibly being an animal suffering from the trouble common in the herd, she was operated on. That night she began to bleed from the wound and in the morning was dead.

"We found on this farm some sweet clover in very bad condition. It had been hauled from a stack in the field and stored in the barn and the cattle had been on it for about four weeks. Feeding of this was discontinued and the trouble ceased shortly afterwards.

"We brought samples of this feed with us and having no calves to try it on we fed it to rabbits. In from three to four weeks' time these rabbits came down manifesting the same symptoms as those manifested by the cattle and showing the same lesions on autopsy. I may say that the observation of all who have done anything with this matter has been that cattle quite uniformly come down on the 28th to the 32nd day after feeding is begun.

"The question Mr. Kerl raises as to who found that sweet clover contained brucin I am unable to answer. I do not recall that I have seen that statement in any of the published reports of this trouble nor did I hear it mentioned by Dr. Roderick. I do not remember having seen this statement in your textbook on poisonous plants, although as I recall it

you do state that cumarin is a poisonous constituent.

"I do not think that brucin, an alkaloid related to strychnin, has been found in sweet clover. These alkaloids occur in Strychnus nux vomica and are powerfully toxic."

WILD FOUR O'CLOCK

Dr. W. T. Bossenberger of Williams, Iowa, sends the following letter:

"In the attached package I am enclosing a sample of weed which I wish to have identified and am also anxious to know whether or not this weed is responsible for the illness of several horses on the farm from which the weed was taken. The afflicted animals showed symptoms of silage poisoning, but as there is no silo on the farm the illness is due to some other causes. They may have eaten some spoiled oats or the weed which I enclose."

This weed proves to be the wild four o'clock (Oxybaphus nyctagineus). It is a well known perennial weed with swollen joints, purplish flowers, and a large thick root. This plant is related to the marvel of Peru, which is commonly cultivated in gardens. I have no record that the wild four o'clock is poisonous, although I have noticed that cattle do not ordinarily eat it.

GOLDEN GLOW

Dr. E. L. Harvey of Geddes, South Dakota, sends a specimen, and the following letter:

"I am sending you today by parcel post a plant which I wish you would identify and tell me whether or not it is poisonous to hogs if eaten in large quantities. This plant grows very plentifully in woods and especially along low spots, to the height of about four or six feet."

The specimen sent is the original of golden glow, or tall cone flower (Rudbeckia laciniata), a tall smooth branching perennial with yellow heads, found in low grounds along streams, common east of the Rocky Mountains to the Missouri River and to the Atlantic coast south to Florida. This plant has been sent to me so frequently as being suspicious, that Dr. Shaffner's statement that it is poisonous is now more or less verified. I have received it from many sources in Iowa and Wisconsin. Dr. J. W. Winter of Norwalk, Iowa, recently sent a specimen of the same plant with the suggestion that it is poisonous. It is well to graze cattle rather sparingly in pastures where it occurs. I have published several accounts of it in these columns.

SNEEZEWEED

Mr. E. H. Hesberg of Farmington, Iowa, writes concerning sneezeweed as follows:

"I am sending to you in a separate envelope some weeds that I would like you to analyze, to see if they hurt sheep and lambs. I have a lot of these weeds in my pasture and my sheep eat the tips off the weeds, and they act duffy and get down on their backs and can't get up. If I get some of them up they will reel around and get all right, but some die."

Sneezeweed is a well known poisonous plant and has long been recognized as such. No doubt owing to the shortage of pasture this year cattle and other live stock are consuming more of these poisonous plants than they otherwise would consume. Concerning the poisonous properties of this weed, I may say:

"Sheep, cattle and horses that are unfamiliar with the plant are often poisoned by it when driven to localities where it is abundant. As a rule these animals avoid it, but it is said they sometimes develop a taste for it and are quickly killed by eating it in large quantity. The poisonous constituent has not been closely investigated, but it is known that it exists principally in the flowers. The young plants appear to be only very slightly dangerous. In the mature ones the amount of poison present seems to vary greatly even in the same field. The symp-

toms as determined by experiments made in Mississippi upon calves, are an accelerated pulse, difficult breathing, staggering, and extreme sensitiveness to the touch. In fatal cases death is preceded by spasms and convulsions.

RAYLESS GOLDENROD CAUSE OF POISONING

That the disease of cattle in the southwestern United States commonly known as "alkali disease" or "milk sickness" is caused by the eating of rayless goldenrod has been proved by experiments carried on by the United States Department of Agriculture. The most serious complaints of this disease are from the Pecos Valley, in the region from Roswell, N. Mex., to Fort Stockton, Tex., where there have been considerable losses of cattle and horses, according to Department Bulletin No. 1391-D, "Rayless Goldenrod as a Poisonous Plant," which contains a report of the experiments.

In the feeding tests cattle were made sick in 11 days by eating 1.5 pounds daily per 100 pounds of animal; sheep were similarly affected by eating daily for about 21 days 1.25 pounds per 100 pounds of animal; while horses were made sick in 18 days on a daily dose of about 1 pound per 100 pounds of animal.

Animals that were fed cut grass or kept in pasture during the experiments were not so readily poisoned as those kept on dry feed. Evidently the grass feeding, presumably because of its laxative effect, tended to delay the production of symptoms and to aid in recovery.

Because of the belief that milk sickness is conveyed to humans by milk or butter, which is probably true, it is almost impossible to sell these products from Pecos Valley farms where the cows are allowed to run on pastures.

Stock eat the rayless goldenrod, it has been observed, only when confined in pastures where there is little good forage. The obvious remedy, therefore, is to see that the animals are well fed. It is practicable also to dig out the weed from fenced pastures.

SNAKE ROOT

Dr. B. F. Ward of Glenwood, writes as follows concerning sickness caused by consuming snake root (Eupatorium urticaefolium):

"I practiced in Will and Grundy counties for 20 years and have seen cattle and people affected with milk sickness. I distinctly remember two horses sick with the trouble, one a mild case, the other I raised in slings, treated them for nephritis and both got better. The main thing is to keep the bowels open. I never believed snake root was the sole cause of the disease, and so told Ferris of a Joliet newspaper, and I think it was a Mr. Marsh. My opinion is all the vegetation is poisonous, a phosphorescent poison exudes from the ground, always a damp shady ground where the sun never shines. Of course, I believe they eat more of the snake root than other vegetation. I believe if the soil was fed in various kinds of feeds it would kill stock and people just the same; that is, the soil in the places where milk sickness occurs, damp, oozing soil is worse; where it is dry and the sun shines on the soil and the snake root grows, it will not kill cattle or horses. At least that is what I observed. The worst places were the Mazon Creek near Morris, Illinois; also, but not quite so bad, the creek running in about five miles east of Morris, Illinois (and a little north), and some down the river (Illinois), some at Wilmington, Illinois, near Custer Park, east side of the river."

Many queries have come to me concerning this plant. Last fall it was reported quite commonly in southwestern and western Iowa. There is no longer any question but that this plant growing in damp woods is injurious. There is nothing to the phosphorescent theory. The plant in shady, damp grounds is more vigorous. It is certainly not true that all vegetation in such damp places is poisonous.

BUFFALO BUR

Dr. F. M. Cowden of Craig, Nebraska, sends a specimen of buffalo bur, with the following letter:

"Find enclosed specimens of a nettle that I would like your opinion on, as to whether the young plant would be poisonous to pigs that eat it. The pastures here are dry and this plant seems to thrive and stay green and tender, and the pigs eat of the tender leaves before the spines are developed on the plant.

"I held postmortem examination on three, finding icteric colored fluid in the peritoneal cavity, greatly distended gall bladder, liver substance, dark and friable with bile secreting tubules filled with thick caseous bile substance. Fat tissue of animal icteric. The animals died suddenly."

Buffalo bur has a yellow flower, spiny leaves, spiny fruit, and certainly is injurious in a mechanical way. Probably also poisonous otherwise, producing the same poisonous substances or glucosides as other species of Solanum. The other plants contain the alkaloid solanin, and solanidin.

The characterstic symptoms are about the same in man and animals. They are stupefaction, staggering, loss of speech, feeling and consciousness, cramps, and sometimes convulsions. The pupil of the eye is generally dilated. Death is directly due to a paralysis of the lungs, but fortunately few cases are fatal.

"Creeping eruption" is a skin affection of the human produced by a larval nematode. This condition is quite prevalent in the southeastern and southern coastal states. It is probable that rats and some of the smaller domestic animals are the hosts of the adult form of the nematode.

State medicine was made a reality by the Veterans' Act of 1924, in which Congress authorized the hospitalization at government expense of disabled veterans regardless of the nature or origin of the disability. This privilege of treatment at government expense was made available to certain classes of veterans regardless of the ability of the individual to provide treatment at his own expense. The House of Representatives passed a bill in May extending this free government service to outpatients, and including women who served as nurses under contract from 1896 to 1901. This bill has not been passed by the Senate. Thus state medicine is infringing on the medical profession, and no doubt unless there is a reaction against such principles and practices, further legislation increasing the scope of state medicine and limiting the field of the private practitioner will be forthcoming.

A NEGLECTED DRUG—CHELONE

Chelone refers to the whole plant of Chelone glabra. This medicinal plant, carrying positive tonic and hepatic properties, is found growing in damp soils in the United States and is known by the common names of balmony, snakehead, turtlehead, salt rheum weed, etc.

Indications and uses: Dyspepsia with hepatic torpor, jaundice and in round and thread worm infestation. In fact, it is an enemy to every kind of worm infesting the animal body.

Therapy: In small and medium doses, this is one of our very best gastro-intestinal tonics. But owing to the fact that the profession has become so accustomed to the use of quinin and arsenical preparations, it has of late been somewhat overlooked. Whenever we are treating dyspepsia or chronic indigestion where there is lack of hepatic secretion, this is a potent remedy. In the malaise and marasmus following distemper in dogs, where quinin has been used freely, chelone is a good remedy and soon restores a tonic condition to the system. In jaundice it also does good work. It is a bitter dose but can be disguised by adding tincture of lavender or cinnamon water.

The plant yields an active principle—

chelonin. It is one of the very few I use, and I have used it many times with good results, not only in expelling worms, but in toning up the intestinal tract so that worms do not find it a desirable location. I have used chelonin as a tonic for young puppies and for the destruction of worms quite frequently, sometimes adding small doses of santonin or podophyllin.

It is one of the very best tonics for the horse, especially for the shaggy yearling with indigestion and filled up with worms. My favorite prescription for this is the following:

℞

Sal carolinum factitum	℥ viii.
Powdered balmony	℥ vi.
Powdered nux vomica	
Powdered juniper, aa	5 iv.

M.

Sig: Tablespoonful in each feed.

This drug is not official in the United States Pharmacopeia. However, this does not lessen its value in my estimation, because it is hard to get around facts. But it is so much handier and less trouble to use the hand-me-down preparations that we almost lose sight of worthwhile remedies that we could prescribe and get results far superior to those from the much used preparations.

G. W. Browning, V. S.

Mobile, Ala.

Even water has its "pathology." Rowntree has described phenomena of what he terms "water intoxication" following introduction of very large amounts of the fluid under certain experimental conditions. In diabetes insipidus, the water balance is set at a level higher than normal. The level can be lowered immediately by the subcutaneous administration of the extract of the posterior lobe of the hypophysis. Subsequent to the use of the extract, the ingestion of water in the large amounts to which the patient has become accustomed results in marked toxicity. Larson, Weir and Rowntree observed patients who developed headaches, nausea, asthenia, incoordination, marked sweating and, in one instance mild subcutaeous edema under these conditions. Patients with chronic nephritis and hypertension who took water in quantities up to 10 liters a day developed headache, dizziness, restlessness, chills, fullness of the abdomen, vomiting, dyspnea and cramps in the legs, marked increase in weight, and increase in blood pressure.—Journal A. M. A.

TAIL CHASING IN DOGS

All animals apparently have objectionable habits and vices, and the canine species is no less susceptible than the equine. In the larger cities where the latter is being crowded out by the encroachment and popularity of motor driven vehicles, veterinary practice flourishes as before, but has taken on a different aspect.

Man apparently must have pets, and with the decline of horse use and the increasing crime wave, the dog is becoming more and more popular, both as a pet and source of protection to its owner.

The habit or vice I have reference to is that commonly termed "chasing the tail."

Case No. 1.—German Shepherd dog, four months old. When brought under our observation, the animal would go round in a circular manner, snapping at its tail for a period of ten to fifteen minutes at a time, and on stopping did not appear dizzy, but acted quite normally. Occasionally he would catch his tail and go round in a lively manner as if on a pivot. It appeared to be just a playful habit of which he seemed never to tire.

We allowed him to associate with other dogs of about his own age in the hope that it might induce him to indulge in more normal recreation, but as soon as the novelty of his new companion wore off, he would resume his old habit.

We next used a local anesthetic at the base of the tail to ascertain if the trouble was due to some abnormal feeling brought about by faulty circulation or improper functioning of the nerves of that particular part of his anatomy, but he carried on as usual.

The next procedure was amputation of

the tail after which he was less active until the wound healed, when he resumed his habit as before, with the exception that he could not catch the stump of the tail with his teeth. The animal was finally destroyed.

Case No. II.—Fox terrier, six months old, with same habit as in the preceding case. Treatment was similar and finally the tail was amputated but without satisfactory results.

Some practitioners claim that as the animal grows older the habit abates. Any advice or discussion on this subject will be appreciated. E. M. Jones.

Buffalo, N. Y.

FOREIGN BODIES IN THE ESOPHAGUS OF DOGS*

Case No. I

On January 23, 1925, a dog was presented for fluoroscopic examination. History showed that about four months before the dog had swallowed a bone and that he showed symptoms of choking for several minutes thereafter. Since that time he had vomited frequently and showed a capricious appetite. He had been treated by several veterinarians for gastritis.

Fluoroscopic examination showed a large bone lodged in the esophagus immediately in front of the diaphragm. Barium monoxid water passed the obstacle freely. A boiled solution passed more slowly and often caused vomiting.

The general condition of the animal being good, it was decided to attempt to expel the foreign body by provoked vomiting. The following treatment was applied at intervals of a few days:

(1) Ingestion of thick soup.
(2) Liquid petrolatum.
(3) Apomorphin.

The result was negative.

On February 2nd another examination was made and the bone found in the same location. On February 6th the dog was operated on as follows:

(1) Anesthesia, 5 mgm. morphia and ¼ mgm. atropin subcutaneously; ⅕ cc. somnifene intravenously.

(2) Cervical esophagotomy—extraction of foreign body under control of fluoroscopic screen by means of a pair of long bullet forceps.

February 10th, slight suppuration.

February 15th, recovery complete.

Case No. II

On March 15th, 1925, a dog was presented with a history that he had been vomiting or trying to vomit for the preceding 48 hours. No gastric pain was revealed on clinical examination. Pulse 130; respiration 32; temperature 39.4° C.

Fluoroscopic examination showed an angular bone in the thoracic esophagus behind the heart. Barium monoxid solution passed slowly above the obstacle only. Operation was decided upon.

(1) Anesthesia, 5 mgm. morphin, ¼ mgm. atropin subcutaneously; chloral hydrate 0.65 in 1-10 solution.

(2) Cervical esophagotomy—extraction of foreign body under control of screen, with forceps.

Three hours after the operation, the subject was awake and walking about. Two hours later he was dead.

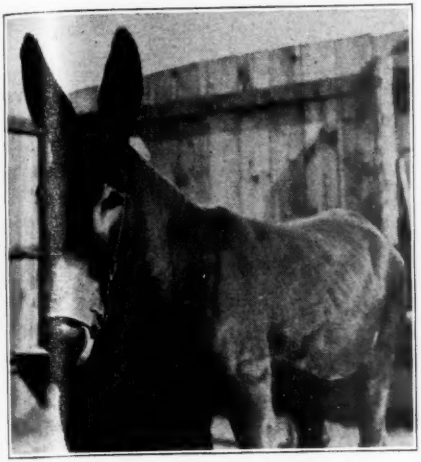
Autopsy showed a subserous abscess of the esophagus. There was a tear through the thickness of the wall. Purulent exudate in the pleural cavity. Marked congestion of the mediastinum. No inflammation of the parietal pleura. The pathology seems to have been as follows: The bone perforated the mucosa and nusculature of the esophagus, causing a subserous abscess. In extracting the bone, the pus was evacuated into the pleural cavity. Nevertheless, this does not seem to explain the cause of death occurring so soon, not preceded by any alarming symptoms.

The cervical route seems to be the best for the extraction of foreign bodies in the thoracic portion of the esophagus, provided the operation is performed under control of a fluoroscope.

*Translated by Captain A. C. Wight, V. C. 12th Field Artillery, Fort Sam Houston, Texas, from article by J. Taskin and Chaussee in Recueil de Medecine Veterinaire, March 30, 1926.

MUFFLER FOR MISSOURI MOCKING BIRD

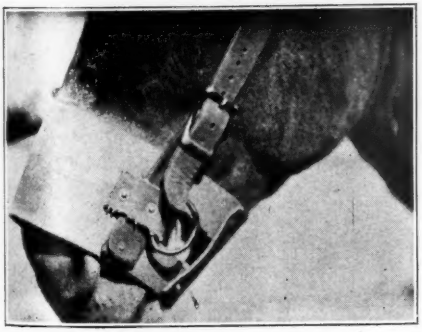
Being annoyed by the persistent vocal exercises of a jack, Dr. H. J. Coggins, of Craigmont, Idaho, devised the muffler as shown in the accompanying illustrations. Practitioners will be doing their communities a wonderful service to rec-



Muffler for Missouri mockingbird

ommend something of this nature to their clients who keep "Missouri mockingbirds."

The nose band is made of 5-inch belting, which fits snugly, but is adjustable by means of two buckles and the strap



Detail of muffler

around the ears. It is placed on the musician's nose every evening.

Instead of just a song at twilight resembling a football rally, the vocal static is held down to a few feeble squeaks which sound like a wild goose dying with the asthma in a blizzard.

"The jack that once through Tara's halls
The soul of music shed,
Now stands as mute in Tara's halls
As though that jack were dead!"

—E. T. Baker.

Moscow, Idaho.

GLEANINGS FROM FOREIGN PERIODICALS*

Tuberculous Iritis in the Cat, by M. Lesbouyries, Recueil de Medecine Veterinaire, Jan. 30, 1926.

After having reviewed the literature of tuberculous eye lesions in domestic animals the author describes two cases of tuberculous iritis in cats which came under his observation.

The first patient was a five-year-old cat, presented because of panting and vomiting. The animal had a double pleurisy. Both eyes were dull. The iris was a dirty yellow and tumefied in the periphery. The contour of the pupil was regular except at one point where a yellowish globular mass the size of a pea obstructed the pupil in part. The iris could still expand and contract. Vision was still present. An ophthalmoscopic examination after use of atropin, 1-200, did not show any lesions in the posterior chamber. The diagnosis of iritis, double, tuberculous, secondary to pleuro-pulmonary lesions was confirmed by the tuberculin test. The tuberculin test consisted of the injection of ¼cc. dilute tuberculin (1-10) subcutaneously. The temperature exceeded 104 degrees.

Another cat showed similar lesions of the right eye. Suspecting tuberculosis, a careful physical examination was made, which was negative. Finally the tuberculin test was administered and the temperature rose from 100.8° F. to 103.5° F. At the sixth hour after the injection of the tuberculin, there was a local reaction

*Review by Capt. A. C. Wight V. C., 12th Field Artillery, Fort Sam Houston, Texas.

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in the affected eye; the cornea and the aqueous humor became cloudy and the iris was very much contracted. There was no lacrymation or photophobia. The next day the eye became transparent. Autopsy showed no miscroscopic lesions other than the eye lesion. Bacteriological examination of the eye tisue was negative. Animal inoculation was positive.

Pathology and Treatment of Ascites in the Dog, by M. Lesbouyries, Recueil de Medecine Veterinaire, Jan. 30, 1926.

The author states that in fifty per cent of cases ascites and tuberculosis were associated. The treatment consisted of the intravenous injection of 10-20 grams of calcium chlorid in a 20-1000 solution. This was given daily and the animal tapped at the beginning of the treatment and later as indicated. In one case the ascites was permanently cured in four days. Another dog was given 10-15 grams per os daily for five days with recovery.

Treatment of Colics of the Horse by Wenger, Schweizer Archiv. March 31, 1925.

There are still numerous obscure points in the diagnosis and treatment of the colics of the horse. Statistics collected by Reinhardt on 125 horses which died of colic show that only about one-fourth were incurable from the onset. One-third of the deaths were due to impaction of the stomach and intestines, and volvolus.

Tschokke states that usually volvulus can be attributed to the falling and rolling of the horses; other causes may be paresis due to stasis of fecal matter or bloating.

Rupture of a stomach containing but little food is often found. It has been noted that rupture of the stomach often follows the administration of physostigmin. This observation is important from the point of view of treatment. Those who consider want of contraction as the principal cause of colics use agents to provoke intestinal peristalsis, while those who attribute colics to too much peristalsis employ narcotics.

The author has treated a large number of cases of colic; using peristaltic stimulants 12 per cent died, while using narcotics (50-80 grams tincture of opium in an infusion of camomile) 7.4 per cent died.

FOOT-AND-MOUTH DISEASE IN GREAT BRITAIN

Outbreaks of foot-and-mouth disease in domestic animals have been giving much trouble in recent years. Last month the number of outbreaks was eighteen; in April, ten, and in March, nineteen. Recently Scotland, which had previously been free, became the center of an outbreak, and in connection with this the important discovery was made that it originated in foreign pigs brought for curing to British bacon factories. There have been seven outbreaks in the vicinity of a bacon factory at Carluke, and they were all traced to virulently infected carcasses brought in from Holland and Belgium through Rotterdam. The virus is supposed to have been conveyed by human agency or by sewage. There has been another case of foreign infection through Carlisle, and a shipload including infected carcasses has arrived at Leith. It has been long suspected that foot-and-mouth disease might be brought to this country by foreign meat, and especially offal, which is obviously an extremely dangerous source of infection. For the first time there is absolutely definite and complete evidence. The government has therefore prohibited the importation of carcasses from the continent. The only exceptions from this prohibition are: (a) fully cured bacon and ham, lard or rendered fat, cooked or preserved meats or meat essences, and (b) hides and skins that have been dried, dry-salted or wet-salted. Wet salted hides or skins must be accompanied by a certificate to the effect that they have been wet-salted for a period of fourteen days.—London correspondent, Journal A. M A.